IMAGING IN SEIZURE PATTERNS

Dissertation submitted to

The Tamil Nadu Dr. M.G.R. Medical University,

Chennai – 600032

With partial fulfilment of the regulations for the

award of Degree

M.D . GENERAL MEDICINE

BRANCH – I

DEPARTMENT OF MEDICINE

K.A.P.V. GOVT. MEDICAL COLLEGE,

TRICHY.

APRIL 2015

BONAFIDE CERTIFICATE

Certified that the dissertation titled **"IMAGING IN SEIZURE PATTERNS"** is a bonafide work done by **Dr GANESH. V,** under my guidance and supervision, in Partial fulfilment of regulations of The Tamil Nadu Dr. MGR Medical University for the award of M.D. Degree Branch I, (General Medicine) during the academic period from May 2012 to April 2015.

Prof .Dr K. PARIMALADEVI M.D.,
Unit Chief MIV
Department of General Medicine
K.A.P.V Government Medical College
Tiruchirappalli.

Prof .Dr. P. KANAGARAJ.M.D.,
Professor & H.O.D
Department of General Medicine
K.A.P.V Government Medical College
Tiruchirappalli.

Prof. Dr. P. KARKUZHALI, M.D.,
The Dean
K.A.P.V Government Medical College
Tiruchirappalli.

DECLARATION

I solemnly declare that the dissertation titled **"IMAGING IN SEIZURE PATTERNS"** was done by me at K.A.P.V Government Medical College, Tiruchirappalli under the guidance and supervision of **Prof. Dr. K. PARIMALADEVI. M.D.,** The dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of the requirement for the award of M.D. Degree in General Medicine.

Place: Tiruchirappalli

Date :

Dr GANESH.V

Postgraduate student

M.D General Medicine

K.A.P.V G M C and

M.G.M G H

K.A.P.VISWANATHAM GOVT. MEDICAL COLLEGE
TIRUCHIRAPALLI -1
INSTITUTIONAL ETHICS COMMITTEE

CERTIFICATE OF CLEARANCE

This is to certify that the project work titled **Imaging studies in seizure patterns** proposed by **Dr.V.Ganesh** part of fulfillment of M.D/M.S course in the subject of **Medicine** for the year **2012-2015** by The Tamilnadu Dr.MGR Medical University has been cleared by the ethics committee.

CHAIRMAN,
Institutional Ethics Committee
K.A.P.Viswanatham Govt. Medical College, Tiruchirapalli -1

ACKNOWLEDGEMENT

I sincerely thank **Prof. Dr. P. KARKUZHALI . M.D (PATH),** Dean,

K.A.P.V Government Medical College, Tiruchirappalli for having permitted me to undertake the study in this prestigious institution.

It is a great pleasure to express my sincere thanks to **Prof. Dr. P.KANAGARAJ, M.D.,** Head of the Department of Medicine for his guidance and encouragement.

With extreme gratitude, I express my indebtedness to my beloved unit Chief Prof **.Dr. K. PARIMALADEVI, M.D.,** for her motivation, guidance and critical suggestions, which enabled me to complete this work.

I am thankful to our medicine chiefs **Prof. Dr. G ANITHA .M.D**., and **Prof. Dr.V. RAJENDRAN M.D** for their valuable guidance and motivation. I am thankful to **Dr. N. SENTHILNATHAN , M.D,** Registrar, Department of Medicine for his valuable guidance and encouragement.

I gratefully acknowledge my indebtedness to **Prof. Dr. R.RAVI, Head of Department, DEPT. OF RADIOLOGY,** who helped me and gave me valuable guidance in doing this research.

I whole heartedly thank **Prof. Dr. M.A ALEEM M.D, D.M (Neuro), HEAD OF DEPARTMENT, NEUROLOGY and Dr.M.RAJASEKAR, M.D,D.M(Neuro) and Dr.E.ARUNRAJ M.D, D.M(Neuro)** for their valuable support and suggestions.

I would like to express my gratitude to ASST. PROFESSORS **Dr. A.SETHURAMAN M.D.** and **Dr S.PALANIVELRAJAN M.D**., of my unit for their constant encouragement, timely help and valuable criticism.

I sincerely thank the Biochemistry Department for their cooperation and support. I thank my post graduate colleagues and CRRI'S for their help and suggestions.

I thank all those patients who participated in this study for their sincere co-operation.

Last but not the least, I thank God.

CONTENTS

ABSTRACT

KEYWORDS – Seizure Disorder, Imaging ,MRI ,Hippocampal volumetry

AIMS AND OBJECTIVES

- *To study the Neuroimaging findings in patients presenting with various patterns of Siezure disorder using Magnetic Resonance Imaging.*
- *To measure the Hippocampal volume in MRI in seizure disorder patients with no structural lesions or any visually detectable changes on routine assessment.*

METHODOLOGY

Materials and Methods

Source of Data

This study was conducted at Mahatma Gandhi Memorial Government Hospital , Trichy in collaboration with Department of Radiology .

Study Design

Descriptive study

Period of Study

January 2014 to September 2014

Ethics Committee Approval

Approval was obtained from Institutional ethics committee.

Inclusion Criteria

- Age > 12
- Documented history of convulsion , who have MRI brain done on them as Out-patient or inpatient
- Consent to the study (patient and /or patient's legal guardian)

Exclusion Criteria

- Age <12
- Diabetic, chronic renal disease, suspected metabolic encephalopathy
- Patients with convulsions with history of acute antecedent events like Trauma, Drugs , toxins, fever .

Consent

An Informed consent was obtained from all the participants and their guardians wherever necessary.

Method

In this study ,56 participants aged >12 presenting with seizure as OUTPATIENT/INPATIENT in Medicine department between January 2014 and September 2014were studied after getting informed consent from patient and /or legal guardian. History taking and clinical examination was done and recorded in the form of a proforma. History included age, sex, duration of seizure, type of seizure, time, any predisposing factors, antecedent events if any, pork ingestion, contact with open case of tuberculosis etc. Detailed ,head to foot, examination including examination for any focal neurological deficit was done. Neuro imaging (mri) was obtained after stabilization.

In those imaging studies where no obvious visually detectable changes were found,hippocampal volumetry was done using these steps:

- Acquisition of MRI slices(coronal) and evaluation in a DICOM viewer(radiant) and exporting them in the form of JPEG image .
- Creating stacks of image slices IMAGE J software.
- Marking REGION OF INTEREST on the stacked image and measuring the area of it.
- Area is the multiplied with the number of slices stacked varying per viewer/and or patient
- Sum of these values per slice is used to calculate volume of 3D structure.
- The acquired data is entered into a MICROSOFT EXCEL sheet and analysed.

Hippocampal Volume calculation

Step 1 ImageJ software(version 1.33) was downloaded from http://www.rsb.info.nih.gov/ij/download.html.

Step 2 Stack creation- Relevant MRI slices were evaluated in the original viewer called RADIANT DICOM VIEWER. The software is downloadable for free. Every MRI slice has a distinctive cipher or number so as to be able to be set up in the information menu of the viewer,which matches a JPEG file. The opened in ImageJ to Create a stack using “Convert Images to Stack” function.

Step 3 Scale adjusting - subsequent to opening DICOM images in ImageJ, the scaling of the images is automatically corrected by the software, and volumetric analysis can be continued. However, in non-DICOM viewers, the scale of the imported stack was adjusted by measuring the distance between two randomly chosen but clearly recognisable points on a slice in the original viewer using its measurement tool. Subsequently, the line between these points was traced on the corresponding slice and its distance set in ImageJ using the "Set scale" function .

Step 4 Region of Interest creation - On the MRI slices, the region of interest (ROI) pertinent for the study at hand is the hippocampus. The region of interest is selected using polygon selection tool with multiple clicks to outline the area.

Step 5 To calculate volume the area of ROI is multiplied by slice thickness for each and added together.giving the total volume of structure.

Marking Region of Interest

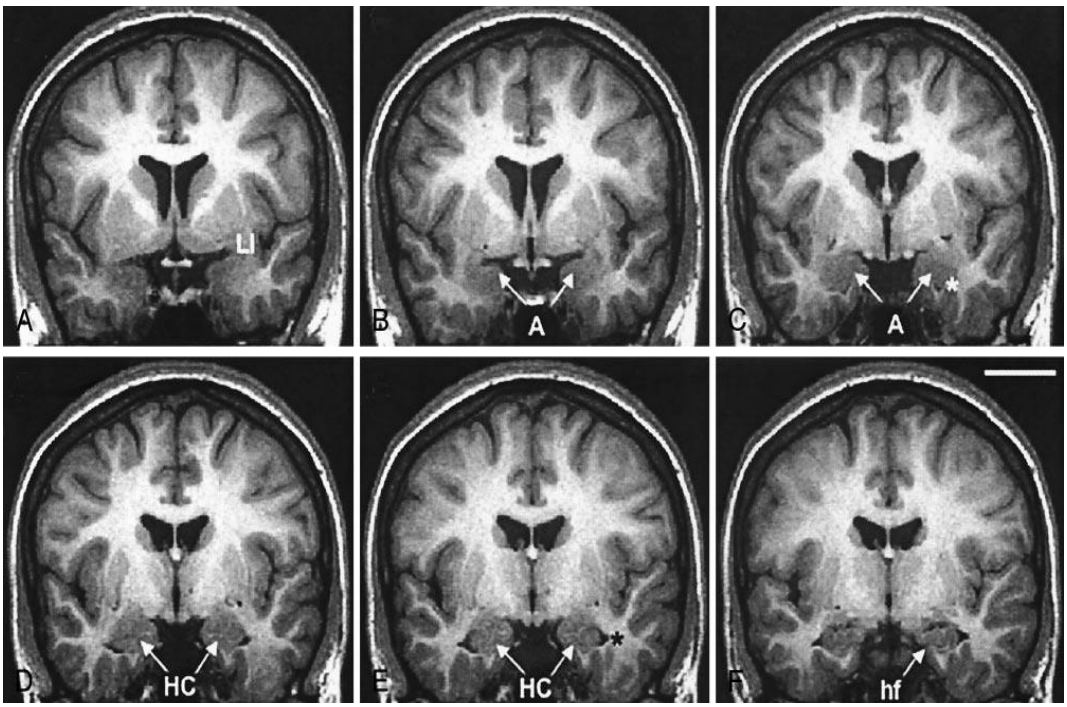


FIGURE A : most rostral where limen insulae is identified(li)

FIGURE B : first section where characteristic oval shaped AMYGDALA(A) is identifiable

FIGURE C : full extent of AMYGDALA where lateral ventricle is seen beneath(*)

FIGURE D : ROSTRAL HIPPOCAMPUS(HC) is seen

FIGURE E : HIPPOCAMPAL HEAD(HC) AND LATERAL VENTRICLE(*)

FIGURE F : HIPPOCAMPAL FISSURE (HF)

Statistical Analysis

Statistical analysis was done by using percentages, mean values, standard deviation, standard error, chi square tests.SPSS version 20 was used to analyse data. The level of significance used was 0.05 levels for the corresponding degree of freedom to draw the inference. A p-value < 0.05 was considered to be statistically significant and a p -value > 0.05 was considered to be not statistically significant.

AIMS AND OBJECTIVES

- *To study the Neuroimaging findings in patients presenting with various patterns of Siezure disorder using Magnetic Resonance Imaging.*
- *To measure the Hippocampal volume in MRI in seizure disorder patients with no structural lesions or any visually detectable changes on routine assessment.*

REVIEW OF LITERATURE

Historical Review

Epilepsy is one of the diseases that has been identified and recorded since the beginning of charted history. It was thought to be spiritual possession state by many including Mesopotamians (2000 BC);Babylonians , Punarvasa atreya(900 BC),and Charaka(400 BC).

Ancient Greeks believed epilepsy to be connected to Intellect and ability and they thought the people affected had superhuman abilities like Hercules and called it the Sacred disease.

Hippocrates in the fifth century BC was the first person to believe it was a disease of the Brain and called it the “great disease” thus the origin of the modern term Grand Mal.

The first anti epileptic medication “bromide” was introduced in mid 1800’s.Phenobarbitone was developed in 1912 and Phenytoin in 1938.

Definition

The word seizure refers to an ephemeral episode of signs and/or symptoms caused owing to peculiarly excessive neuronal action of the cerebral cortex**.**

Epilepsy is a state defined by recurring, unprovoked episodes of seizure. The implication of the expression *seizure* needs to be carefully delineated from that of epilepsy. *Epilepsy* refers to a circumstance in

which a person has seizures which recur due to a persistent, underlying development. This definition implies that a person with a solitary seizure, or recurrent seizures due to correctable or preventable circumstances, does not essentially have epilepsy. Epilepsy refers to a clinical trend rather than a solitary disease entity, since there are many forms and causes of epilepsy. However, among the many causes of epilepsy there are various *epilepsy syndromes* in which the medical and pathologic characteristics are unique and suggest an explicit underlying aetiology. though a diversity of factors affect the occurrence and frequency of seizures, 5–10% of the populace will have at least 1 seizure, with the highest incidence occurring in early youth and middle age.

Using the definition of epilepsy as two or more unprovoked seizures, the incidence of epilepsy is 0.3–0.5% in different populations throughout the world, and the prevalence of epilepsy has been estimated at 5–10 persons per 1000[8].

The identification of epilepsy is time and again not uncomplicated, and misdiagnosis is common [1].

A thorough and dependable description of the occurrence by an observer is critical to the conclusion and assessment, which may perhaps not be accessible.

The rationale of the investigative assessment in a patient with seizure is to offer substantiation that help prove or disprove the identification of epilepsy and to discover the reason of epilepsy and/or to categorize the epileptic disorder.

Neuroimaging has a significant part in the assessment of patients with refractory epilepsy for Surgical management .The medical management in approximately 15% to 40% of patients receiving Antiepileptic drug therapy, remains inadequate due to ongoing seizures or undesirable side effects [2,3,4].

Some of them can be possible candidate for surgery. The choice of surgery depends on seizure type and anatomic substrate, among other factors. For surgical resection or disconnection to be offered,

a) The seizure must be focal in origin
b) Accurate preoperative localization of the epileptogenic focus must be available.

The most favorable candidates for surgery are those with complex partial seizures and a unilateral temporal lobe focus, in whom rates of cure and significant improvement approach 90 percent in some series, but overall, are probably closer to 50 percent after 5 years.

A randomized trial conducted by Wiebe and colleagues gave representative results after temporal lobectomy of 58 percent of 40 carefully studied patients remaining seizure-free after 1 year, in contrast to 8 percent on medication alone[5].

Furthermore, as reported by Yoon and colleagues, among those patients who remain free of seizures for 1 year after surgery, over half are still free of seizures after 10 years and most of the remainder had one or fewer episodes per year[6].

The clinical presentation of epilepsy is varied, and it is necessary to categorize them according to established classification schemes to select appropriate workup, therapy, and to assign prognosis. The most widely used classification of epileptic *seizures* is the International League Against Epilepsy (ILAE) classification[7]

INTERNATIONAL CLASSIFICATION OF EPILEPTIC SEIZURES

I.Generalized seizures (symmetrical and with no local onset)

A. Tonic, clonic, or tonic-clonic (grand mal)

B. Absence (petit mal)

1.With lapse of consciousness

2.Complex with brief tonic, clonic, or automatism

C. Lennox_Gastaut syndrome

D. Juvenile myoclonus(JME)

E. Infantile spasms (West syndrome)

F. Akinetic (astatic, atonic) seizures (with myoclonus)

II.Partial, or focal, seizures

A. Simple (devoid of loc or change in psych)

1. Motor frontal lobe (tonic, clonic, tonic-clonic; jacksonian march; benign childhood epilepsy; epilepsia partialis continua)
2. Somato-sensory or special -sensory (visual, auditory, olfactory, gustatory, vertiginous)
3. Autonomous functions.
4. Pure extrasensory

B. Complex (with diminished consciousness)

1. Commencement as plain partial seizures and continuing to diminished sensorium
2. With altered sensorium at inception

III. Special epileptic syndromes

A. Myoclonus and seizures

B. Reflex epilepsy

C. Acquired aphasia with convulsion

D. Febrile seizures

E. Conversion reaction

CLINICAL TYPES

Generalized Seizures

The primary generalized epilepsies are a group of somewhat varied, age- dependent phenotypes that are characterized by global 2.5- to 4-Hz bifrontally predominant spikes or polyspike-and-slow-wave discharges that arise with no fundamental structural abnormalities. In most instances, these individuals have normal intellect. What is most significant is that a genetic component underlies many of these disorders . By contrast, seizures that begin locally and progress into generalized tonic- clonic seizures, termed secondary generalized seizures, generally have no such genetic component and are usually the result of causal brain disease, either acquired or due to inherited malformations or metabolic defects. Quite often, the initial focal phase is missed, foremost to misdiagnosis. Individuals with secondary generalized epilepsies tend to have more diffuse brain dysfunction and may have a progressive course.

These seizures may be of varying types, including atonic, myoclonic, and tonic-clonic seizures. An escalating frequency and severity of this group of disorders with age reflects the accretion of focal insults from trauma, strokes, and other injury.

The patient sometimes senses the approach of a seizure by one of numerous skewed phenomena (an aura). For some time maybe hours, the patient may sense lethargic , dejected, petulant, or, very rarely, ecstatic. myoclonic jerks on arousing might portent a seizure later in the day. In one in two cases, there is various form of movement for some time before unconsciousness (head turning and lateral gaze), even though the patient fails to form a recollection of this and such information is obtained only from an onlooker. Abdominal pains or cramps, a plummeting, mounting, or riveting sensation in the Epigastric region, paleness or flush of the face, pounding headache, Bowel movements have also been given significance as a prodrome, but These lack consistency and are not enough to be supportive.

Most often, the seizure strikes without forewarning, Starting with a sudden unconsciuosness and drop to the floor. The motor signs include a concise flexion of body, an open mouth and eyelids, and upgazing eyes. The upper limbs are elevated and abducted, the elbows semiflexed, and the hands pronated. Then a more lingering extension (tonic) phase, involves the back, neck, then the upper and lower limbs. A high pitched cry is common due to expiration of air through a closed glottis. Since the muscles of respiration are caught up in the tonic contraction, breath is suspended, and some moments later cyanosis may develop. pupils are

dilated , unreactive to light. Micturition is common at this stage or during post ictal stage. tonic phase lasts for a few seconds.

Clonic phase begins when, At first there is a meek indiscriminate shudder, due to cyclic reduction of tonic spasm. It begins at a rate of eight per second and coarsens to four per second; then it hastily leads to brief, flexor spasms which occur as periodic salvos and disturb the whole body. The face becomes flushed and distorted by a sequence of grimaces, and often tongue bite. Autonomic signs are important: the pulse is rapid, blood pressure is elevated, pupils are dilated, and salivation and sweating are profuse; bladder pressure may increase sixfold during this phase. The clonic jerks reduce in frequency and extent over a time of 30 s. The subject remains breathless till the conclusion of the clonic stage, which is noticeable by a profound inspiration. Instead of this whole vivid sequence described above, the seizures may be shortened or limited in scope by anticonvulsive medications.

In the last phase of the paroxysm, all actions have come to an end and the Subject lies immobile and flaccid, in a deep loss of consciousness. The pupils contract to light. Respiration may be silent or stertorous. This state persists for more than a few minutes, after which the subject opens his eyes to stare on and is noticeably bewildered , perplexed and restless. The subject might converse and later be amnesic for what he said. he becomes lethargic and falls asleep, sometimes for

several hours, then sometimes awakens with a throbbing headache. When completely improved, has no recollection of any the spell but knows something has occured ; the evident apprehension of those around him; and a tender, bitten tongue and hurting muscles . The latter, if violent enough, may lead to a compressed vertebral body or result in a serious harm; a fracture, periorbital hemorrhages, SDH, or burn may be sustained Each of these phases of the generalized tonic-clonic seizure has its characteristic EEG accompaniment. Initially, movement artifacts obscure the EEG changes; sometimes there are cyclic spikes or spike-wave discharges lasting a few seconds, followed by an approximately 10-s period of 10-Hz spikes. As the clonic phase asserts itself, the spikes become diverse with slow waves and then the EEG slowly assumes a polyspike-and-wave pattern. When all actions have ceased, the EEG tracing is nearly flat for a variable time, and then the brain waves resume their preseizure pattern. convulsion of this type in common come piecemeal or in groups and occur when the subject is wakeful and active or asleep , or frequently when falling asleep or awakening. 5 to 8 percentage of patients will at various period have a long-lasting succession of such seizures without consciousness between them; called status epilepticus and requires critical management. Sometimes first outburst of seizures takes the form of convulsive status.

Few clinical states closely simulate a grand mal convulsion, but several are worthy of mention. One is a clonic jerking of the extended limbs (usually less severe than those of a grand mal seizure) that occurs with vasodepressor syncope or a Stokes-Adams attack. In contrast to an epileptic type of EEG, the brain waves are slow and flat during the jerking movements.

A rarer phenomenon that may be indistinguishable from a generalized convulsion occurs as part of basilar artery occlusion (9). This apparently has its origin in ischemia of the corticospinal tracts in the pons; a similar ischemic mechanism in the cortex has been invoked for limb- shaking TIA(transient ischemic attacks), in which there are clonic actions of one limb or one side of the body throughout an event of cerebral ischemia. In infants, a breath-holding spell may resemble the tonic phase of a generalized seizure.

Idiopathic Nonconvulsive Seizures (Absence, Petit Mal)

In distinction to Grand mal seizures, absence seizures (formerly petit mal or pykno-epilepsy) are noteworthy for briefness and the lack of motor action. they may be so short-lived that the subjects are, from time to time, not aware of them; to a spectator, they bear a resemblance to a tick of inattentiveness or pensiveness. The event, coming without admonition, consists of a abrupt break of consciousness, for which

French word “absence”(not present or not in attendance•) has been retained.

The patient gives a blank stare and momentarily ceases to react. about 10 percent of such patients are entirely motionless during the attack; in the remainder, one observes a brief burst of fine clonic movement of the eyelids, face, or finger or synchronous movement of both upper limbs at a rate of 3/sec. This corresponds to the EEG irregularity, which takes the type of a generalized three- per-second spike-and-wave pattern . Minor automatisms in the form of lip-smacking, chewing, and fumbling movements are not uncommon at some point in an event but do not assume importance. Postural tone may be to some extent reduced or elevated, and infrequently there is a minimal vasomotor disarray. such patients never fall; they may even persist such complex tasks like walking or cycling. After 2 to 10 s, infrequently longer, the subjects get in touch with the surroundings and resumes activity. Only a failure of the continuity of the activity betrays the occurrence of the blank period (the absence). In many such patients, intentionally hyperventilating for 3 minutes is an useful way to induce absence attacks.

Typical absence seizures comprise the most distinctive epilepsy of Childhood ; hardly ever do the seizures commence before 4 yrs of age or ater than teenage years . a further trait is their immense rate of recurrence. As many as hundreds may come about in a day, at times in bursts at definite times of day. Generally they relate to periods of lack of concentration and may appear when the child is sitting silently rather than absorbed in his classes. If numerous, they can disturb attention and thought to the point so as to hamper the child's performance in school . Such attacks can last hours with no gap of normal rational activity between them ,thus called absence or petit mal status. Small, faint three-per-second myoclonic movements are the only motor exhibit (myoclonic petit mal), and are accompanied by a continuous three-per-second spike-wave defect in the EEG. Most cases of absence status have been described in adults with frontal lobe epilepsy.

Such attacks may begin or end with GTCS or a burst of seizures. Absence may well be the lone kind of seizure in infancy. The events tend to reduce in occurrence in puberty and then often disappear, only to be replaced in several instances by major generalized seizures.

Absence Variants

To be notable from archetypal absence seizures are varieties in which the lapse of awareness is not as much of total or myoclonus will be

predominant, and other in which the EEG aberrations are not often of a 3-per-second spike-and-wave type (they could occur at the rate of 2 to .5/sec or of 4- to 6-Hertz polyspike-and-wave complex). Atypical petit mal is a term that was coined to illustrate protracted runs of slow spike-and-wave activity, usually with no noticeable loss of consciousness. External stimuli such as asking the patient to answer a question or to count will disrupt the run of abnormal EEG activity.

About one in three children affected by absence attack will, present with symmetrical or asymmetrical myoclonic jerks devoid of lapse of awareness, and about one in two will at some point in time have major generalized (tonic-clonic) convulsion.

A common and somewhat benevolent variety of myoclonic seizure occur in late youth and teens (JME).

In contrast to the aforesaid epilepsies is a variety that has its ensues between 2 and 6 years of age and is comprised by atonic, or astatic, seizures (i.e., falling attacks), often succeeded by a mixture of combination of minor motor, tonic-clonic, and partial seizures and by progressive cerebral impairment in connection with a unique, slow (1-2Hertz) spike-and-wave pattern. This is Lennox-Gastaut syndrome. Often presents in earlier life by infantile spasms, a typical EEG picture (3-Hz hypsarrhythmias •), and an pause in mental maturity, a trio occasionally referred to as the West syndrome . The early commencement

of atonic seizures with falls, injuries, and related abnormality nearly constantly has a grave implication. Prematurity, perinatal injury, and metabolic diseases of infancy are the most common underlying conditions. This is essentially symptomatic generalized epilepsy, in distinction to the former idiopathic types. The Lennox-Gastaut syndrome may continue into adult years and is one of the largely complicated forms of epilepsy to treat.

The notion that absence, myoclonic, and akinetic seizures constitute a petit mal triad, as formerly proposed by Lennox, has been by and large discarded . Akinesia (motionlessness) is not unique to any type of seizure.

The classic absence, with or without myoclonic jerks, hardly ever cause the Subject to fall and have to be grouped under a distinct entity due to its Relatively benign nature.

Partial or Focal Seizures

The International Classification divide all seizure into two groups - generalized, focal or partial (more recently termed localization-related), partial seizures differ with the background of the injury and usually divided into 2 , simple and complex Simple partial seizures most frequently begin from foci in the sensorimotor cortex. Complex partial seizures largely often have their focus in the temporal lobe on one side or

the other, but a frontal localization is also well recognized. The sites of the aberrant lesions and the types of seizures to which they give rise are listed below. These relations are so useful in diagnosis, they should be well-known to all physicians.

Regional localisation of seizures(10)

Clinical type	**Localization**
Somatic motor	
Jacksonian march	Prerolandic gyrus
Masticatory, salivation, speech arrest	Amygdaloid nuclei, opercular
plain contraversive	Frontal
Head turning with arm movement or athetoid-dystonic posturing	Supplementary motor cortex
Somatic and special sensory (auras)	
Somato-sensory	postrolandic
amorphous images, illumination, pattern	Occipitalcortex
Auditory	Heschl's gyri
Vertiginous	Superior temporal
Olfactory	Mesial temporal
Gustatory	Insula
instinctive: autonomic	Insular-orbital-frontal cortex
Complex partial seizures	

Formed hallucinations	Temporal neocortex or amygdalohippocampal complex
Illusions	
Dyscognitive experiences (deja vu, absent-minded states, depersonalized)	
emotional states (fear, depression, or elation)	Temporal lobe
Automatisms	Temporal and frontal lobe
Absence	Frontal lobe, amygdale,hippocampi, reticular-activating system
Bilateral myoclonic	Reticulo-cortical, fronto-central

Partial Seizures arising in frontal lobe(Focal Motor and Jacksonian Seizures) Focal or partial motor seizures are attributable to a discharging lesion of the contralateral frontal lobe. The most frequent type, originating in the auxiliary motor area, takes the form of a turning motion of the head and eyes to the opposite side , often associated with a tonic contraction of the trunk and limbs on same side. This may comprise the whole seizure, or it may be followed by widespread clonic movements; the extension of the seizure may occur immediately before or concurrently with loss of consciousness. On the other hand, a lesion in one frontal lobe may give rise to a major generalized convulsion without an initial turning of the head and eyes. It has been postulated that in both

types of convulsion, the one with and the one without turning movements, there is an instant spread of the discharge from the frontal lobe to integrating centres in the thalamic or high midbrain reticular formation, accounting for the loss of perception. Seizures that begin with vigorous, persistent deviation of the head and eyes, and every so often of the entire body, are referred to as versive or adversive. Since the turning movements are typically to the side opposite the lesion (sometimes to the same side), contraversive and ipsiversive, respectively, would be preferable terms. Nonforceful, non sustained, or apparently indiscriminate lateral head movements during the ictus do not have localizing value.

Contraversive deviation of only the head and eyes can be induced most constantly by electrical stimulation of the superolateral frontal region (area 8), just anterior to area 6. Less consistently, the same movements can be obtained by stimulating the more anterior portions of the frontal cortex, or the auxiliary motor area, and the temporal or occipital cortex seemingly through extend of the ictus to the frontal contraversive area. In temporal lobe epilepsy, early in the seizure, there may be head turning ipsilaterally followed by vigorous, contraversive head (and body) turning. These head and body movements, if they occur, are preceded by silent staring and other automatisms. The Jacksonian motor seizure begin with a tonic contraction fingers of one arm, the face ,then the muscles of foot. This changes into clonic movements in these

parts in a pattern similar to that in a GTCS. Sometimes a series of clonic movements of escalating frequency build up to a tonic contraction. The movements may loiter limited to a small area or widen from the area first affected to other muscle groups on the ipsilateral side . In the latter, or classic, jacksonian form, which is comparatively rare, the seizure spreads from the hand , up the arm, to the face, and down the leg; or, if the first movement is in the foot, the seizure marches up the leg, down the arm, and to the face, usually in a matter of 20 to 30 s. Interestingly, spontaneously occurring focal motor seizures, e.g., those beginning in the toes or fingers, may sometimes be inhibited by applying a ligature above the affected region or, in the case of focal sensory seizures, by applying a forceful sensory stimulus in front of the advancing sensory aura. infrequently, the first muscular contraction is in the abdomen, thorax, or neck. In some cases, the one-sided seizure activity is followed by turning of the head and eyes to the convulsing side, occasionally to the contralateral side, and then by a generalized seizure with unconsciousness. Consciousness is not lost if the sensorimotor symptoms remain limited to one side.

Following convulsions that have a major focal motor signature, there may be a short-lived paralysis of the affected limbs. This Todd's paralysis persists for minutes or at times for hours after the seizure, usually in proportion to the period of the convulsion. persistent focal

paralysis beyond this time usually indicates the existence of a focal brain lesion as the primary cause of the seizure. A similar event is found in cases of focal epilepsy that involve the language, somesthetic, or visual areas; here the persistent deficit corresponds to the region of brain afflicted. high frequency of commencement of focal motor epilepsy in the face, hands, and toes is probably related to the inexplicably large cortical representation of these parts. The disease process or focus of excitation is usually in or in close proximity to the rolandic (motor) cortex, i.e., area 4 of Brodmann ; in some cases, and particularly if there is a sensory adjunct, it has been found in the postrolandic convolution.

Lesions restricted to the motor cortex are reported to assume the type of clonic contractions, and those confined to the premotor cortex (area 6), tonic contractions of the contralateral arm, face, neck, or all of one side of the body. Tonic elevation and extension of the contralateral arm (fencer's posture) and choreoathetotic and dystonic postures have been associated with high medial frontal lesions (area 8 and supplementary motor cortex), as have composite, bizarre, and flailing movements of a contralateral limb, but this always raises the suspicion of conversion reaction. Perspiration and piloerection occur occasionally in parts of the body involved in a focal motor seizure, suggesting that these autonomic functions have a cortical representation in or adjoining to the rolandic area. Focal motor and Jacksonian seizures have essentially the

same localizing implication.

Seizure discharges arising from the cortical language areas may give rise to a short aphasic deficit(ictal aphasia) and ejaculation of a word, or, more commonly, a vocal arrest. Ictal aphasia is usually succeeded by other focal or generalized seizure activity but may occur in segregation, without loss of consciousness, in which case it can later be described by the patient. Postictal aphasia is more frequent and has much the same localizing value. Vocalization at the onset of a seizure has no such importance. These disturbances should be distinguished from the stereotyped repetition of words or phrases or the muddled speech that characterizes some cases of complex partial seizures or the postictal confusional state. As pointed out by Manford and colleagues[12], relatively few focal seizures can be localized precisely from clinical information alone. However, when combined with scalp and intracranial EEG recording and MRI, the data are convincingly accurate.

Somato-sensory, Visual, and Other Types of Sensory Seizures

Somatosensory seizures, focal unilateral, are almost always indicative of a focus in or in close proximity to the postrolandic convolution of the opposite cerebral hemisphere. Penfield and Kristiansen found the seizure focus in the postcentral or precentral convolution in 49 of 55 such cases(13). The sensory disorder is usually described as lack of

sensation, tickle, or a pins-and-needles feeling and uncommonly as a feeling of creeping (formication), current, or movement of the part. Pain and thermal sensations can arise but are extremely rare. In common , the commencement of the sensory seizure is in the oral cavity, digits and the spread to neighbouring parts of the body following a pattern determined by sensory arrangements in the postcentral (postrolandic) convolution of the parietal lobe. If the sensory symptoms are localized to the head, the focus is in or nearby to the lowest part of the convolution, near the sylvian fissure; if the symptoms are in the leg or foot, the upper part of the convolution, near the superior sagittal sinus or on the medial surface of the hemisphere, is involved.

Visual seizures are comparatively rare but also have localizing implication. According to Gowers[14], red is the most frequently reported color, followed by blue, green, and yellow. These images may be referred to the visual field on the contralateral side of the lesion or may appear directly ahead. If they occur on one side of the visual field, patients make out that only one eye is affected (the one opposite the lesion), probably because most persons are aware of only the temporal half of a homonymous field deficiency. Curiously, a seizure arising in one occipital lobe may cause brief blindness in both fields. It has been noted that lesions on the lateral surface of the occipital lobe (Brodmann's areas 18 and 19) are likely to cause a sensation of flash or pulsating lights.

More intricate or formed visual hallucinations are more often than not due to a focus in the dorsal part of the temporal lobe, near its junction with the occipital lobe, and may be associated with auditory hallucinations.

The localizing value of visual auras has been confirmed recently by Bien and colleagues[15] in a study done on surgically treated patients with intractable seizures. They found that simple visual hallucinations and visual loss were classic of occipital lobe epilepsy but could also occur with seizure foci in the anteromedial temporal and occipitotemporal regions.Auditory hallucinations are uncommon as an initial expression of a seizure. Occasionally a patient with a focus in one superior temporal convolution will describe a buzzing or roaring in the ears. A human voice, sometimes repeating unrecognizable expression, or the sound of music, has been noted a minority of times with lesions in the more posterior part of one temporal lobe. Vertiginous sensations of a type indicative of a vestibular source may on rare occasions be the first warning sign of a seizure. The lesion is usually located in the superoposterior temporal region or the intersection between parietal and temporal lobes.

In one of the cases reported by Penfield and Jasper[10], a sense of vertigo was evoked by stimulating the cortex at the junction of the parietal and occipital lobes. Occasionally with a temporal focus, the

vertigo is followed by an auditory feeling. faintness, or light-headedness, is a frequent preface to a seizure, but this symptom, has so many diverse connotations that it is of petite diagnostic value. Olfactory hallucinations are frequently linked with disease of the inferior and medial parts of the temporal lobe, generally in the region of the parahippocampal onvolution or the uncus (hence Jackson's term uncinate seizures). Usually the seeming odor is exteriorized, i.e., projected to someplace in the environment, and is described as unpleasant or rank, though otherwise unidentifiable. Gustatory hallucinations have also been recorded in established cases of temporal lobe illness and with lesions of the insula and parietal operculum; salivation and a feeling of thirst may be related. Electrical stimulation in the depths of the sylvian fissure, extending into the insular region, has formed peculiar sensations of taste. indistinct and frequently beyond description visceral sensations arising in the thorax, epigastrium, and abdomen are amongst the most common of auras.

Most often they have a temporal lobe origin, even though in several such cases the seizure discharge has been localized to the upper bank of the sylvian fissure; in a few others, the focus was located in the upper or middle frontal gyrus or in the medial frontal area near the cingulate gyrus. Palpitation and increase of rate of the pulse at the onset of the attack have also been associated to a temporal lobe focus.

Complex Partial Seizures (Psychomotor Seizures, Temporal Lobe Seizures) These vary from the major generalized and absence seizures (1) the aura may be either a simple focal seizure type or hallucination or perceptual misapprehension, indicating (frequently) a temporal lobe derivation (2) instead of a complete loss of Awareness , there is a period of istorted behavior and perception, for which the patient is later found to be amnesic. Although it is difficult to enumerate all the psychic experiences that may occur during complex partial seizures, they may be categorized into a somewhat subjective hierarchy of illusions, hallucinations, dyscognitive states, and affective experiences. Sensory illusions, or distortions of ongoing perceptions, are the most common. Objects or persons in the environment may shrivel or retreat into the distance, or they may expand (micropsia and macropsia), or perseverate as the head is moved (palinopsia). slanting of the visual surroundings has been reported.

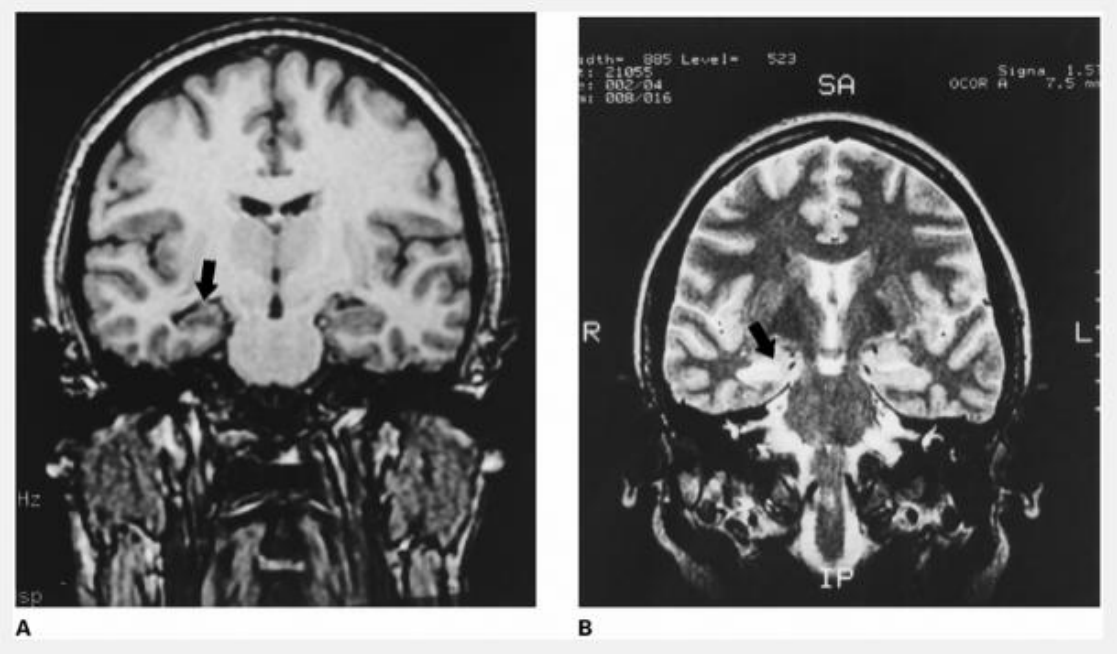
Hallucinations are most often visual or auditory, consisting of formed or unformed visual images, sounds, and voices; less frequently, they may be olfactory (usually unpleasant, unidentifiable sensations of smell), gustatory, or vertiginous. The term dyscognitive state refers to feelings of increased reality or familiarity (deja vu) or of incongruity or unfamiliarity (jamais vu) or a sense of depersonalization. Fragments of certain old memories or scenes may insert themselves into the patient's

mind and recur with striking clarity, or there may be an sudden pause of memory. Associated epigastric and abdominal sensations have been alluded to above. Emotional experiences, while less common, may be dramatic sadness, loneliness, anger, happiness, and sexual excitement have all been recorded. Fear and anxiety are the most common affective experiences, while occasionally the patient describes a feeling of rage or severe anger as part of a complex partial seizure. Ictal fear may have no clear connection to objective experience and is usually not related to the situation in which the patient finds himself during the seizure. The patient with temporal lobe seizures may show evidence of only one of the foregoing manifestations of seizure activity or various combinations of them. In a study done by by Lennox[16], 43 percent displayed some of the motor changes; 32 percent, automatic behavior; and 25 percent, alterations in psychic function. Because of the numerous agreement of these symptom complexes, he referred to them as the psychomotor triad.

Probably the clinical outline varies with the precise locality of the lesion and the course and extent of spread of the electrical discharge. Because of their focal origin and complex symptomatology, all these types of seizures are best subsumed under the title of complex partial seizures. This term is preferable to temporal lobe seizures, since typical complex partial seizures sometimes arise from a focus in the medial-

orbital part of the frontal lobe. Also, seizures originating in the parietal or occipital lobes may be manifested as complex partial seizures because of seizure spread into the temporal lobes. Often the brief ictal aura is not reflected in cortical epileptic activity and therefore may be missed by routine surface EEG recordings. Complex partial seizures are not peculiar to any period of life, but they do show an increased incidence in adolescence and the adult years. Ounsted and co-workers[17] concluded that , about one-third of such cases could be traced to the occurrence of severe febrile convulsions in early life . As a corollary, about 5 percent of all their patients with febrile seizures continued to have seizures during adolescence and adult life; in the latter group there were many in whom he seizures were of the temporal lobe type. Neonatal convulsions, head trauma, and various other nonprogressive perinatal neurologic disorders are antecedents that place a child at risk of developing complex partial seizures [18].

Two-thirds of patients with complex partial seizures also have generalized tonic-clonic seizures or have had them at some earlier time, and it has been theorized that the generalized seizures may have led to secondary ischemic damage to the hippocampal portions of the temporal lobes. In the latter cases, carefully performed and quantitated MRI in the coronal plane may disclose a loss of volume in the hippocampi and adjacent gyri on one or both sidesi.e., medial temporal sclerosis.



Medial temporal sclerosis. A. Shrunken right hippocampus (shown by arrow) and secondary enlargement of lateral ventricle. B. T2-weighted image showing signal change in the hippocampi (shown by arrow).

The Nature of the Discharging Lesion

Physiologically, an abrupt alteration of CNS function onsequential from a convulsive high-frequency or high voltage low frequency discharge. This discharge arises from an group of excitable neurons in any part of the cerebral cortex and possibly in secondarily involved subcortical structures as well. Of course, there need not be a visible lesion. In the proper conditions, a seizure discharge can be initiated in an entirely normal cerebral cortex, as when the cortex is activated by ingestion or injection of drugs, by withdrawal from alcohol or other

sedative drugs, or by repeated stimulation with subconvulsive electrical pulses (kindling phenomenon). Viewed from a larger physiologic viewpoint, seizures need 3 conditions: (1) a populace of pathologically volatile neurons; (2) an augmented glutaminergic activity through recurring associations in order to spread the impulse (3) a reduced activity of inhibitory GABA-nergic neurons.

The last of these has been challenged, but it is supported by considerable data and serves as a reasonable model, as noted below. Understanding of the initial discharges and their spread has been greatly advanced by the identification of several rare forms of familial epilepsy that are the direct result of mutations in sodium, potassium, acetylcholine receptor, or GABA channels on neurons. Just why the neurons in or near a focal cortical lesion discharge abnormally is not fully understood. Some of the electrical properties of a cortical epileptogenic focus suggest that its neurons have been deafferented. Such neurons are known to be hyperexcitable, and they may remain so chronically, in a state of partial depolarization, able to fire irregularly at rates as high as 700 to 1000 per second. The cytoplasmic membranes of such cells appear to have an increased ionic permeability, which renders them susceptible to activation by hyperthermia, hypoxia, hypoglycemia, hypocalcemia, and hyponatremia as well as by repeated sensory (e.g., photic) stimulation and

during certain phases of sleep (where hypersynchrony of neurons is known to occur).

Pathology of Epilepsy

In most autopsied cases of primary generalized epilepsy of the grand mal and absence types, the CNS has been said to be grossly and microscopically normal. However, it is improbable that the brains in these cases were examined totally at least there is not a single case that has been subjected to whole-brain serial sectioning in a search for disorders of neural migration and old scars. Not unexpectedly, there are also no visible lesions in the seizure states complicating drug intoxication and withdrawal, transient hyper- and hyponatremia, and hyper- and hypoglycemia, which presumably correspond to derangements at the cellular level. In contrast, most of the so-called secondary epilepsies have definable focii. These include zone of neuronal tissue loss such as a porencephaly, heterotopia, dysgenetic cortex, hamartoma, vascular malformation, and tumor.

The frequency of these lesions is not fully known. Certainly the focal epilepsies are associated with the highest incidence of structural abnormalities, although in certain cases no morphologic change is visible. In several series of cases of temporal lobe excisions, a specific pattern of neuronal loss with gliosis (sclerosis) in the hippocampal and amygdaloid region was found in the majority[19], and this abnormality is being

increasingly recognized with MRI. Vascular malformations, hamartomas, and low-grade astrocytomas were less frequent; in a small number, no abnormalities could be found. The extensive use of CT and MRI represents an important surrogate approach to the pathologic study of epilepsy. More than 25 years ago, Gastaut and Gastaut reported that in primary grand mal and absence epilepsies, CT abnormalities were found in approximately 10 percent of cases, whereas in the Lennox-Gastaut syndrome, the West syndrome, and partial complex epilepsies it was found in 52, 77, and 63 percent, respectively[20]. Atrophy, calcification, and malformations were the most recurrent changes. MRI and predominantly the FLAIR images have proved to be a particularly sensitive means of detecting epileptogenic lesions of the medial-basal portion of the temporal lobes . Repeatedly, patients are observed in whom MRI disclosed a cortical or subcortical developmental malformation such as a cortical heterotopia or another surgically treatable lesion of the temporal lobe, even after CT scanning had failed to do so.

More subtle epileptogenic foci may be demonstrated by PET or by interictal SPECT. SPECT done in Ictal stage, which shows hyperperfusion of the seizure focus, is a more demanding but also more sensitive and specific procedure. With mention to the focal epilepsies, it has not been promising to determine which component of the lesion is accountable for the seizures. Gliosis, fibrosis, vascularization, and

meningocerebral cicatrix have all been incriminated, but they are found in nonepileptic foci as well. The Scheibels' Golgi studies[21] of neurons from epileptic foci in the temporal lobe showed distortions of dendrites, loss of dendritic spines, and disorientation of neurons near the scars, but these findings have dubious status since they were not usually compared with similar nonepileptic lesions.

Moreover, changes such as these have proved to be nonspecific and artifactual. Once a gliotic focus of whatever cause, bordered by groups of discharging neurons, becomes epileptogenic, it may remain so throughout the patient's lifetime. Nevertheless, with the passage of years, seizures tend to diminish or cease in as many as half of childhood and adult traumatic epilepsies.The largely common histologic finding in the brains of epileptics is a bilateral loss of neurons in the CA1 segment (Sommer sector) of the pyramidal cell layer of the hippocampus, extending into adjacent regions of both the pyramidal layer and the underlying dentate gyrus. It is still undecided whether this neuronal loss is primary or secondary and, if the latter, whether it was incurred at birth or happened later as the consequence of recurrent seizures.

The cessation of seizures in many patients following medial temporal lobe resection favors the first interpretation . Attesting to the uncertainty of cause or effect regarding hippocampal damage are case reports and surgical series too numerous to list that favor one view or the

other. It can be assured, however, that seizures, even in adulthood, are capable of inducing hippocampal shrinkage. This does not preclude a causative role for medial temporal sclerosis [22].

Computerised Tomography in Epilepsy

Computerised axial tomography is frequently ordered in people presenting with new onset Seizure to an emergency department. It is in general accessible quickly in that setting and can rule out acute neurologic problems that necessitate imperative interference. on the other hand, in non emergent situation, MRI is more Sensitive than CAT and is the imaging[45,46] of choice . It is usually restricted to the emergency department setting in the evaluation of potential acute symptomatic seizures. The Electroencephalogram in Epilepsy The EEG provides confirmation of Hughlings Jackson's concept of epilepsy that it represents a recurring, rapid, disproportionate discharge of cortical neurons. The EEG is unquestionably the most sensitive, indeed indispensable, tool for the diagnosis of epilepsy; but like other ancillary tests, it must be used in conjunction with clinical data.

In patients with idiopathic generalized seizures and in a high proportion of their relatives, interictal spike-and-wave abnormalities without any clinical seizure activity are common, especially if the EEG is repeated several times. inversely, a proportion of epileptic patients have a

perfectly normal interictal EEG; occasionally, using standard methods of scalp recording, the EEG may even be normal during a simple or complex partial seizure. Furthermore, a small number of healthy persons (approximately 2 to 3 percent) show paroxysmal EEG abnormalities; some of them have history of epilepsy running in their family (particularly of absence seizures) and may themselves later develop seizures.

One consistent observation has been that the area of initial spike activity corresponds best to the epileptogenic focus. This rule guides epilepsy surgery. The postseizure or postictal state following generalized seizures also has its EEG correlate, taking the form of haphazard generalized slow waves. Following partial or focal seizures, the EEG shows focal slowing. With clinical recovery, the EEG returns to normal or to the preseizure state. A single EEG tracing obtained during the interictal state is abnormal to some degree in 30 to 50 percent of epileptic patients; this figure rises to 60 to 70 percent if patients are subjected to three or more studies utilizing standard activating measures (hyperventilation, photic stimulation, and sleep).

With structural lesions, focal slow and sharp activity, which is not clearly epileptiform, may be the only clue to a seizure focus. A higher yield of abnormalities and a more precise definition of seizure types can be obtained by the use of everal special EEG procedures. Overnight EEG

recording is particularly helpful because focal abnormalities, particularly in the temporal lobes, may become prominent in stage II sleep. Sphenoidal leads have been used to detect inferomedial temporal seizure activity, but they are uncomfortable and probably add little more information than can be obtained by the placement of additional subtemporal scalp electrodes. In our experience, nasopharyngeal electrode recordings are too contaminated by artifact to be clinically useful.

Activating procedures such as hyperventilation, photic stroboscopic stimulation, and sleep increase the yield of EEG recordings

MRI in Evaluation of Seizure

The majority patients alleged of having had an epileptic convulsion should have a neuroimaging done. The intention is to make out a structural etiology for Epilepsy. The core of elective neuroimaging is magnetic resonance imaging (MRI), which is more sensitive than CAT for most epileptogenic lesions [45,46,47].Exceptions to the need for neuroimaging include children with simple febrile seizures and children whose clinical history and EEG are consistent with benign partial epilepsy of childhood or idiopathic generalized epilepsy.

Specific pathologies — Structural causes of epilepsy that can be identified by MRI include [48,49]:

- Mesial temporal sclerosis (hippocampal sclerosis)
- Malformations of cortical development
- Brain tumors
- Vascular malformations
- Cerebral infarction, cerebral hemorrhage
- Traumatic brain injury

Infections, including encephalitis, cerebral abscess, granulomas, and cysts (neurocysticercosis) Mesial temporal sclerosis, also known as hippocampal sclerosis is a most frequently diagnosed focal structural defect in patients with epilepsy. While most cases present in childhood, it is not uncommon for this disorder to first appear in young adults. Surgical treatment is often curative in patients who do not become seizure-free on medication. MRI characteristics include hippocampal atrophy and increased T2 and FLAIR signal intensity .

Malformations of cortical development (eg, focal cortical dysplasia) are the second most common structural etiology for epilepsy [50,51]. As neuroimaging has become more sophisticated, these are being identified in a greater number of epilepsy patients. MRI is more sensitive

for focal cortical dysplasias with more severe pathologic grade than for milder lesions, and these are more amenable to surgical cure [50].

Subtle findings of focal cortical thickening may be missed on initial interpretation of the MRI and identified when the study is re-reviewed [51]. These lesions are congenital, and related epilepsy usually presents in childhood. However, it is not rare for epilepsy to first develop in young adults. Brain tumors and cerebrovascular disease are more common in older patients. Certain infections, especially neurocysticercosis caused by Taenia solium, are common etiologies of epilepsy in endemic populations. When neurocysticercosis is suspected, MRI with contrast is useful for identifying cysts and evaluating disease activity. However, computed tomography (CT) may add to the diagnostic evaluation, as CT is more sensitive than MRI for detecting small areas of calcification.

Sensitivity — Most individuals with new-onset epilepsy will not have a structural lesion on MRI; in one case series, the yield was 14 percent [48]. Among reported studies, a wide range (1 to 57 percent) of euroimaging studies in patients with epilepsy are abnormal [47]. These differences reflect the technology used (eg, CT versus MRI), the patient

population studied, and the range of MRI abnormalities included as abnormal. As an example, emergency departmentbased studies include a

larger number with acute symptomatic seizures that are more likely to have corresponding CT or MRI abnormalities. Also, older patient populations are more likely to have structural brain lesions identified on MRI as a cause for epilepsy than are populations of primarily younger adults. While brain MRI is routinely used in the clinical evaluation of epilepsy, a substantial gap between the sensitivity and specificity is present for MRI in different care settings [9,16]. In one study, The original MRI interpretation was compared to a reinterpretation of the original MRI and also to the results of a repeat MRI performed using a dedicated epilepsy protocol. The sensitivity of MRI for focal lesions for each of these was 39, 50, and 91 percent respectively.

This was a select patient population of patients with refractory epilepsy that excluded patients with acute ocal brain conditions, including cerebral abscess and rapidly expanding brain tumors. In most cases, the missed diagnosis was hippocampal sclerosis.

Epilepsy protocol for MRI — To optimize the yield, MRI should be performed using an epilepsy protocol. While epilepsy protocols vary depending on the institution and available technology, most recommendations agree that an epilepsy protocol for MRI should ideally include:

- Traumatic brain injury
- Infections, including encephalitis, cerebral abscess, granulomas, and cysts (neurocysticercosis)
- Standard T1-weighted images.
- T2-weighted fast spin-echo sequences. Gradient echo (T2) sequences.Sensitive for small hemosiderin deposits, these may detect smaller Cavernous malformations or prior traumatic brain injury.
- Fluid-attenuated inversion recovery (FLAIR) sequences.

3D volume acquisition sequences with high definition of the gray-white junction These help identify cortical dysplasia and allow 3D reconstruction and volumetrics Imaging sequences should consist of contiguous, thin (<1.5 mm) slices . All the above sequences should be obtained in two orthogonal planes, with coronal images obtained obliquely . The oblique coronal orientation minimizes partial volume effects that otherwise is commonly difficult to understand in hippocampal sclerosis and minute lesions in the temporal lobe. The use of gadolinium is not required for initial diagnostic MRI studies, but can be used to better define pathologies seen on noncontrast study or to improve sensitivity in initially-negative studies [46].

Advanced MRI techniques — Sensitivity also appears to be improved by more advanced MRI technologies that are not universally available at present. One study found that the use of phased-array surface coil MRI performed at 3- Tesla MRI detected focal lesions in 65 percent of patients with a previously negative MRI . Findings included cortical dysplasia, benign tumors, and hippocampal sclerosis. In one-third of patients with a prior abnormal MRI, the 3-Tesla MRI study further characterized the lesion's pathology and anatomic extent. Other reports confirm the superiority of higher field strength imaging study [15].

Susceptibility weighted MR uses the magnetic property of iron in blood has been shown to be significantly more sensitive in detecting cavernous malformations [29]. This technique also appears to be helpful in identifying epileptogenic, postinfectious, calcified lesions (eg, cryptococcus, tuberculosis, cysticercosis) [49]. High-resolution MRI is required for the diagnosis of many malformations of cortical development. On a standard MRI, findings suggestive of dysplasia include cortical thickening, blurring of the gray-white margin, increased signal on FLAIR, and subtle tapering bands of gray matter extending from the cortex towards the ventricles [49]. Subtle lesions can be missed, and false positive MRI readings can result from over-interpretation of normal variations in cortical thickness [51].

Specialized MRI techniques such as magnetization transfer imaging [25], diffusion tensor imaging [51], voxel-based analysis [48], and texture analysis [51] improve detection of malformations of cortical development as well as MRI's ability to detect the full extent of the malformation. The latter is important because complete removal correlates with successful remission of seizures after surgery [34].

Specificity of MRI findings for epilepsy — Not all MRI abnormalities are associated with epileptic seizures many cystic lesions (arachnoid cysts, choroidal fissure cysts), lacunar strokes, ventricular asymmetry, diffuse atrophy, and isolated venous anomalies (ie, those not associated with arteriovenous malformation or cavernous angioma) are not known to be epileptogenic, and should be considered incidental to a seizure diagnosis [49].

When a potentially epileptogenic structural abnormality is seen on brain MRI, it suggests an anatomic substrate for epilepsy, and provides support for a diagnosis of epilepsy. However, such findings should not be interpreted in isolation and must be linked with the subject's convulsion history and electroencephalogram . Several observations suggest that suchfindings might be incidental in a small proportion of patients. Another potential source of diagnostic confusion is that some patients may have acute MRI changes that are attributed to the effect of seizures

rather than their cause. These are more common after prolonged seizures or clusters of seizures, and are characterized by local swelling, increased T2 signal intensity, restricted diffusion, and focal parenchymal and/or leptomeningeal contrast enhancement that resolve on subsequent imaging studies. These are discussed separately.

Functional Magnetic Resonance Imaging

Functional MRI (fMRI) can detect focal changes in blood flow and oxygenation levels that occur when an area of the brain is activated, measured on MRI as the blood-oxygen-level-dependent (BOLD) effect. fMRI can be used to noninvasively map motor, sensory, and language functions, and is most commonly used as part of surgical planning to redict and limit postoperative neurologic deficits, particularly language function [49]. fMRI may eventually replace the carotid amobarbital (Wada) test, particularly for language lateralization [51]. It may eventually assist decision-making and planning of epilepsy surgery[31]

Interpretation of fMRI requires caution; it is an indirect measure of brain function. Discrepancies with the Wada test have been described [51]. Its sensitivity and specificity are imperfect [49] percent compared to the ada test

POSITRON EMISSION TOMOGRAPHY — 2-[18F] fluoro-2-deoxy-d-glucose positron emission tomography (FDG-PET) images the opographic distribution of glucose uptake in the brain and provides a picture of cerebral metabolism. Ictal scans can be useful but are difficult to obtain except in rare cases such as epilepsia partialis continua. It is generally performed as part of a presurgical evaluation.PET is highly sensitive in detecting MTLE FDG-PET can be helpful in lateralizing the epileptogenic temporal lobe in "MRI negative" cases, with a yield that ranges from 45 to almost 90 percent [79-83,88-91].There is less information available regarding the usefulness of FDG-PET in extratemporal epilepsy, but it appears somewhat less sensitive in these cases [49,51]. Another limitation of PET is that the area of hypometabolism typically extends beyond the epileptogenic zone, making it less useful for precise neuroanatomic localization [49]. The use of other tracers (eg, [11C] flumazenil, fluoroethyl-l-tyrosine, alpha methyl tryptophan, and serotonin agonists) is under investigation and holds promise for improving the sensitivity and specificity of PET for presurgical localization [49,51,31]. Similarly, coregistration of FDG-PET and MRI show potential for improved sensitivity and specificity compared with either technology alone [51].

Single Photon Emission Computed Tomography-

In a single photon emission computed tomography (SPECT) study, a radiolabeled tracer, eg, 99mTc-hexamethyl-propyleneamineoxime (99mTc-HMPAO), is injected and binds on first-pass through the brain. Thus, a SPECT study provides a snapshot of cerebral circulation at the time of injection. The tracer is stable for several hours, allowing delayed imaging of blood flow at the time of injection. In patients with focal epilepsy, ictal SPECT studies typically show hyperperfusion at the seizure focus with surrounding hypoperfusion; postictal and interictal SPECT shows regional hypoperfusion [51]. The sensitivity of SPECT is improved by comparison of ictal and interictal SPECT studies with quantitative subtraction techniques or by statistical comparison with a control database (statistical parametric mapping) [51].

Coregistration with MRI, a technique known as subtraction ictal SPECT scan coregistered with MRI (SISCOM) improves localization and can help predict surgical success [51]. Ictal SPECT studies have a high yield in the evaluation of temporal lobe epilepsy that exceeds 90 percent; but the sensitivity for extratemporal seizure foci is lower [31].

Magnetoencephalography and Magnetic Source Imaging

Magnetoencephalography (MEG) is the recording of magnetic fields generated by intraneuronal electrical currents. Magnetic source imaging (MSI) is the combination of MEG source localization with coregistered anatomical imaging (MRI in most cases), in which the magnetic dipole representing an epileptiform discharge is placed on the patient's MRI scan [51]. MEG is similar to EEG. However, while the electrical currents that are measured with EEG are attenuated in strength and spatially blurred by tissues between the brain and the scalp surface, the magnetic fields assessed by MEG are not significantly affected by intervening tissue layers [49]. As a result, MEG may allow for more clinically reliable localization of brain activity . MEG and EEG can be viewed as complementary studies . MEG is maximally sensitive to dipoles situated tangentially to the surface, whereas EEG is maximally sensitive to radial dipoles . Averaged MEG spikes can thus be helpful in detecting discharges that are hidden in the background noise in simultaneous EEG recordings. This phenomenon is reciprocal: in some cases, EEG spikes are more apparent than on MEG. Some studies suggest that MEG has specific advantages for spike detection in extratemporal epilepsies, particularly those that lie superficially on the brain's surface . MEG/MSI has been approved for presurgical localization of epilepsy and may be particularly useful for localization of

spike sources [43].

MEG is also approved for the localization of neuronal function (similar to evoked potentials and functional MRI) for language, sensorimotor, or visual cortex, and has been used to localize other cortical functions as well. MEG language mapping has been shown to agree with results of the Wada test in 75 to 95 percent of patients . Specific lesions

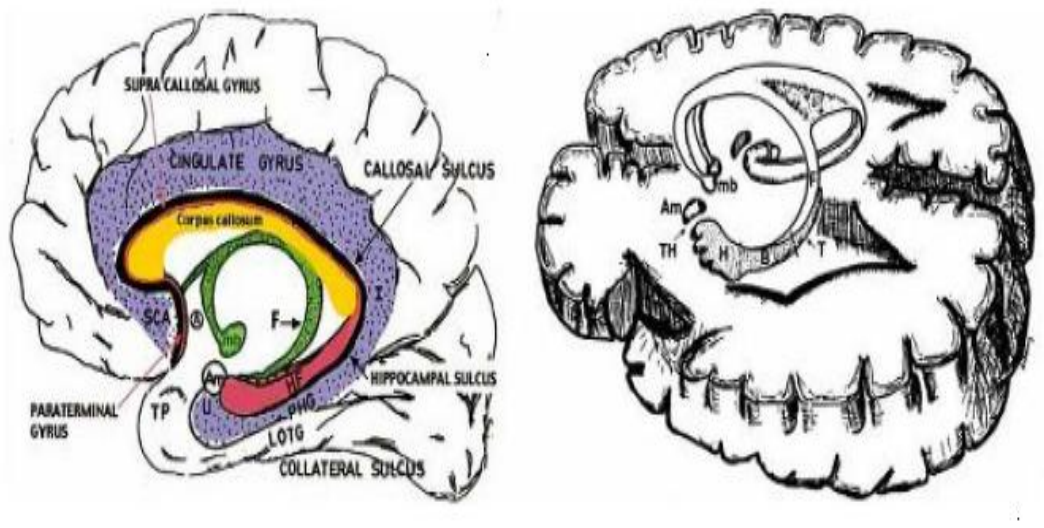
Hippocampal Sclerosis

Mesial temporal sclerosis or hippocampal sclerosis is a extremely epileptogenic aberration associated with temporal lobe complex partial seizures. It is the most widespread entity Encountered in patients undergoing epilepsy surgery. These patients frequently have a history of complex childhood febrile seizures, and onset of recurring medically intractable seizures during the first decade of life. However, MR evidence of hippocampal sclerosis has been established in medically controlled subjects with CPS.

Hippocampus- anatomy

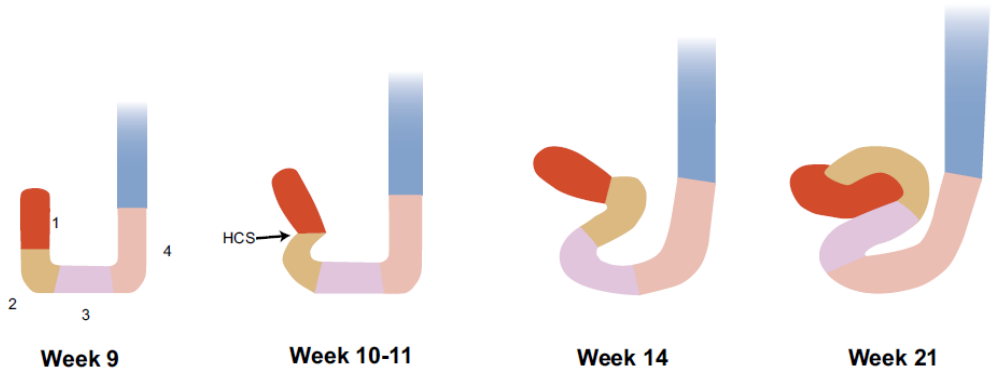
The hippocampus is a convex formation resting on medial portion of temporal lobe consisting of compound U-shaped layers of the dentate gyrus and cornu ammonis, which are interlocked collectively. The ippocampus lies rostral to subiculum and parahippocampal gyrus, to form

a 4 to 4.5cm long arched ridge in the floor of temporal horn of lateral ventricle. Three regions of the hippocampus can be definite based on morphology and relationship to the midbrain. The most anterior expanded part, the hippocampal head or pes hippocampus, can be recognized by three to four digitations on its superior surface. The cylindrically shaped body extends dorsally in the region of the midbrain, and the terminal section—the tail of hippocampus—rapidly narrows behind brainstem. The rounded ventricular surface is covered with ependyma, beneath which tangential white-matter tracts, called alveus, pass medial to congregate to make up the fimbria, which venture into the ventricular cavity and extends as fornix.

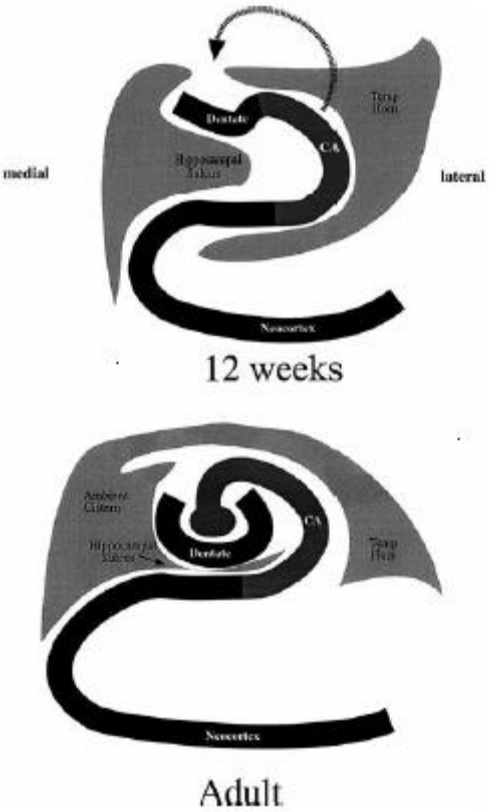


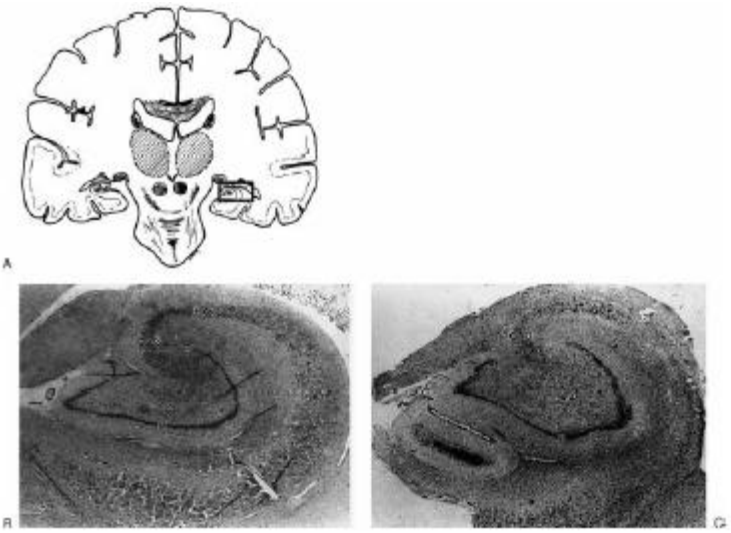
Hippocampal Embryology

At 13to 14 weeks -the unfolded hippocampus is positioned on the medial facade of the temporal lobe and overlay a extensively open hippocampal sulcus. 15 to 16 weeks, the dentate gyrus and cornu ammonis have in full swing to infold, but the hippocampal sulcus remains open. The parahippocampal gyrus is outsized and more medially located. The CA1, CA2, and CA3 fields of the cornu ammonis are arranged linearly (unlike their adult archlike configuration). The dentate gyrus has a slender U shape. By 18 to 20 weeks, the hippocampus begin to look like the adult hippocampus. The dentate gyrus and cornu ammonis have folded into the temporal lobe, with the cornu ammonis and dentate gyrus each form an interconnected arch. The hippocampus and subiculum approximate each other transversely through a narrow hippocampal sulcus. Disorders arising from aberrations of hippocampal development include an unfolded (vertical) hippocampal configuration and hippocampal sulcal cyst remnants due to incomplete closure of the hippocampal sulcus.



Schematic diagram of the fetal development of the hippocampal formation.
1 = Dentate gyrus, 2 = Cornu ammonis, 3 = Subiculum,
4 = Parahippocampal gyrus, HCS = HippoCampal Sulcus.





Hippocampal histology. A: The black rectangle in this diagram of a coronal section through the temporal lobe shows the region of interest in panels B and C. Coronal histologic sections of the hippocampus using Nissl stain (original magnification, 16× to 18×) demonstrate normal histology (B) and hippocampal sclerosis (C). Note the loss of the pyramidal cells in CA1 and CA4 regions of the hippocampus (*black dots*) in the specimen with hippocampal sclerosis. CA, cornu ammonis; D, dentate gyrus. (From Bronen RA, Cheung G, Charles JT, et al. Imaging findings in hippocampal sclerosis: correlation with pathology. *AJNR Am J Neuroradiol* 1991;12:933-940, with permission.)

[25] The MR imaging of amygdala and hippocampus is best performed in a slightly oblique coronal plane, perpendicular to the long axis of hippocampus . The amygdala remains rostral to temporal horn and can be visualised separate from hippocampus by CSF signal in uncal recess of temporal horn . When there is paucity of CSF in uncal recess, the alveus may help delineate these structures. For volumetric assessment of the hippocampus and amygdala, the criteria used most often for defining anatomic boundaries are those described by Watson et al. , with the posterior hippocampal boundary extending to the crus of the fornix. For the particular volumetric technique used at a center, nomograms of hippocampal volume need to be established using data derived from

normal subjects, normalizing for variables including head size, age, sex, and hemispheric side. Hippocampi are isointense to gray matter on all MRI pulse series, may be slightly hyperintense to gray matter on fluid attenuated inversion recovery (FLAIR) images [26] .The prime MR abnormality in Hippocampal scleroses are hyperintensity on T2-W images and/or Hippocampal atrophy[27,28,29,30,31]

Magnetic Resonance Features of Hippocampal Sclerosis

Principal hippocampal findings

- Hippocampus atrophy
- Signal alteration
- Alteration of internal architecture

Secondary findings

- Loss of hippocampal digitations in pes hippocampus
- Dilated temporal horn
- Atrophy of temporal lobe
- white matter atrophy
- Anterior temporal white matter changes
- Formix atrophy
- Atrophy of mammilary body
- Atrophy of thalamus and caudate nucleus

MR has provided invaluable insight into the pathogenesis of hippocampal sclerosis. MRimaging findings help support the "two-hit hypothesis" [32]. This hypothesis proposes that two factors are necessary for hippocampal sclerosis to occur: first an initial precipitating injury (such as a complicated febrile seizure or encephalitis) and second an increased vulnerability (such as a genetic disposition or developmental anomaly). There is MRI evidence to support both acquired (i.e., the precipitating injury) and developmental (i.e., the increased vulnerability) etiologies. Many lines of evidence support an acquired etiopathogenesis.

A temporal sequence of changes has been reported after prolonged febrile and non febrile seizures—the hippocampus initially enlarges with prolongation of T2 relaxation time and then subsequently becomes atrophied[33,34] Quantitative hippocampus volume estimation is established to some extent add to the sensitivity more than plain visual analysis in recognition of sclerosis of Hippocampus [34,36,37,38]. Qualitative assessment of hippocampal atrophy by an experienced observer achieves a sensitivity of 80% to 90%, whereas quantitative methods are about 90% to 95% accurate. Quantitative methods may be of value in large epilepsy surgery centers and centers with limited observer experience. Hippocampal volumetry and T2 relaxometry are routinely useful in diagnosis of bilateral hippocampal atrophy without visually appreciable signal changes. Bilateral hippocampal atrophy occurs in about 10% to 20% of

cases and is frequently associated with developmental anomalies of temporal lobe [39.40]. T2 relaxometry and FLAIR techniques are useful in detecting associated abnormalities of amygdala not seen on routine MR [38].

Detection of associated involvement of amygdala is important because seizure-free surgical outcome is significantly better in isolated hippocampal atrophy (80% vs. 50%) [36]. Quantitative methods have great value in research, allowing the investigator to test hypotheses correlating anatomic data with clinical and pathologic indices. Hippocampal volume from quantitative MR has been correlated with cell loss, frequency of childhood febrile seizures, memory functions, and successful surgical outcome [41,42,43]. Several studies have correlated hippocampal volume loss with duration of epileptic disorder. One study correlated recurrent temporal lobe seizures with hippocampal volume loss, whereas generalized seizures were linked to progressive neuronal damage, as deduced from the association of frequent generalized seizures with metabolic derangement in temporal lobes on MR spectroscopy [44].

Early intervention for seizure control may therefore be indicated to prevent progressive brain damage from recurrent seizure activity [44]. Although quantitative MR imaging has great benefits for research, the everyday clinical appliance of quantitative MR volumetry has a number of drawbacks, such as operator's time; the want for committed personnel,

workstations, and the software; and the requirement of a truly representative data sample of normal control subjects. A word on epilepsy surgery For surgical resection or disconnection to be offered, the seizure must be focal in origin, and accurate preoperative localization of the epileptogenic focus must be available. Localization of the epileptogenic focus is, therefore, the major task in preoperative evaluation of surgical candidates. In the past, EEG was essentially the only method of localizing the seizure focus. Accuracy of the conventional scalp .With regard to refractory complex partial seizures, anterio temporal lobectomy and selective amgdalohippocampactomy are the most commonly performed neurosurgical procedures. The surgical resection can only be performed unilaterally because of unacceptable neurologic consequences of bilateral temporal lobectomy. Therefore preoperative localization and assessment of laterality of seizure focus must be carried out. The algorithm for localization of seizure focus and assessment of resectability varies according to institutional practice The general goals of neuroimaging in presurgical evaluation of epilepsy patients include (i) delineation of structural and functional abnormality in the putative epileptogenic region, (ii) categorization into a specific epilepsy substrate (iii) detection of additional abnormalities (iv) mapping of sensorimotor, language, and memory functions in the epileptogenic and adjacent regions of the brain. Procedures for epilepsy surgery include anterior

temporal lobectomy,lesionectomy,non lesional cortical resection,corpus callosectomy, hemisperectomy.

Neurocysticercosis

For the confirmation of diagnosis of NCC,Neuroimaging is required. Carpio has proposed a classification system [52]that corresponds to the viability of the parasite: Active stage - rounded hypodensity on CT with CSF like signal on MR,both CT and MRI show an eccentric mural nodule(invaginated scolex) when multiple is pathognomonic of NCC called STARRY SKY APPEARANCE; Transitional stage – diffuse hypodensity and irregular borders on contrast CT , low signal intensity ares on T2W MRI images; Inactive – calfied nodule of hyperdensity on CT , Hypointensity on MRI.

Tuberculoma

Comprise of central caseous necrosis enclosed by a zone of peripheral inflammatory cell infiltrate. It is a union of multiple tubercles. Predominance of inflammatory infiltrate is seen in early stages. MRI

Findings

- Hypointensity on T2W images.
- Central iso and hyperintensity on T1W images.
- Central caseation is best seen on T2W images.

Magnetic Resonance Features of Malformations of Cortical Development

- Cortical thickening
- Hyperintensity of gray matter
- Irregularity of gray–white matter junction
- Macrogyria
- Paucity of gyri (pachygyria)
- Polymicrogyria (multiple small gyri)
- Altered sulcal morphology
- Radial bands
- Heterotopic gray matter, ependymal or subcortical
- Transmantle heterotopia
- Homogeneous hyperintense signal in subcortical white matter on T2-weighted images
- Hemispheric enlargement
- Developemental abnormalities – classification

1. Abnormal neuronal and glial proliferation or apoptosis

a) Absent or decreased proliferation like microcephaly,microlissencephaly.

b) Increased proliferation like gangliocytoma, ganglioglioma, hemimegalencephaly.

2. Abnormal neuronal migration

    a. Cobblestone complex

    b. Heterotopia

    c. Lissencephaly

3. Abnormal cortical organization

    a. Schizencephaly

    b. Polymicrogyria

4. Abnormal cortical development

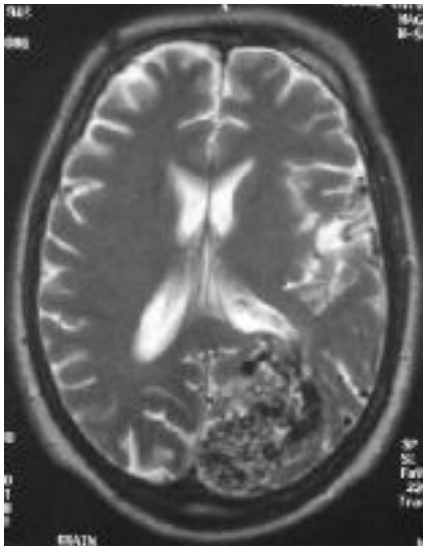
    a. Bilateral glutaric aciduria

    b. Mitochondrial metabolic disorder

    c. Sublobar dysplasia

Arteriovenous malformations

On MRI, a archetypal Arteriovenous malformations appear as a compactly crammed or baggy interweave of vessel .



AVM of the brain.brisk blood surge in the course of engorged vessels cause a signal or current void on regular SE T1- and T2-w image. This finding is exclusively distinctive of AV malformations. MRI can demonstrate the defect dimension , the prime supply of the malformation and venous emptying. MRI can additionally demonstrate connected aneurysms on arterial feeders and associated sequelae, such as cerebral edema ,ischemia or mass effects.

Vascular steal in the brain or spinal cord adjoining to the aberrancy can be visualized as a regional abnormal reduced signal intensity on T1-w images and augmented intensity on T2-w, proton density—w, and short-tau inversion recovery (STIR) image. MR can be used to assess ruptured AV malformation. Hemorrhage in acute stage appears iso-intense on T1-w images , hypo-intense on T2-w image due to presence of reduced hemoglobin in extravasate but intact red blood cells. In sub acute stage, appears hyper-intense on both T1W and T2W image. Chronic stage is seen as a innermost hyper-intense nucleus delimited by a ring of hypo-intensity due to hemosiderin deposit in macrophages in the adjacent parenchyma. Hemosiderin is a little hypo-intense on T1-w images and strikingly hypo-intense on T2-w image.

MR is an exceptional pre-operative preparation implement for distinguish the relation between an AVMalformation and crucial brain structure. the relation between hemispheric AVM and well-expressed

brain region will be clear, mainly with f- MRI. allied aneurysms can be visualised within a hematoma as a void in flow. sadly, the sensitivity of MR to detect aneurysms < 1-2 cm is little[64] .

Studies pertaining to quantitative Hippocampal volumetry

Paramdeep Singh et al. In his study of quantitative and qualitative hippocampal volumetry said that there is no significant difference in volume of right and left hippocampi and quatitative analysis is better for bilateral pathology[65] .

Raz et al ,in his study has said that there is significant hippocampal shrinkage associated with aging[66]. on the contrary, Bigler et al has come up with the conclusion that there is no difference in size with aging in hippocampus[67].

Elisabeth Wenger et al[68] has found that there were differences in Hippocampal Volumetry in the young and old when an automated software (freesurfer was used)

Jaba LS et al, found that there was no difference in volumetric analysis of hippocampus when done by manual method and by using automated software (Image j)[69] .

Pedro M. Gonc et al have stated that ,Volumetric analysis piriform cortex and amygdale may lead to effective localization of seizure focus in patients with image negative epilepsy[70] .

Patrick Kwan,and Martin J. Brodie [72,73] have done an epidemiological study in which they suggest that a set of patients presenting with recurrent epilepsy (20 or more) and in patients with poor control with antiepileptics there an inate defect the blood brain barrier due to which antiepileptics even when given in higher doses cannot fully exert the desired pharmacologic effect and these people have a tendency to encounter AED induced neurotoxicity as higher doses are given to them to control their seizures.

METHODOLOGY

Materials and Methods

Source of Data

This study was conducted at Mahatma Gandhi Memorial Government Hospital , Trichy in collaboration with Department of Radiology .

Study Design

Descriptive study

Period of Study

January 2014 to September 2014

Ethics Committee Approval

Approval was obtained from Institutional ethics committee.

Inclusion Criteria

- Age > 12
- Documented history of convulsion , who have MRI brain done on them as Out-patient or inpatient
- Consent to the study (patient and /or patient's legal guardian)

Exclusion Criteria

- Age <12
- Diabetic, chronic renal disease, suspected metabolic encephalopathy

- Patients with convulsions with history of acute antecedent events like Trauma, Drugs , toxins, fever .

Consent

An Informed consent was obtained from all the participants and their guardians wherever necessary.

Method

In this study ,56 participants aged >12 presenting with seizure as OUTPATIENT/INPATIENT in Medicine department between January 2014 and September 2014were studied after getting informed consent from patient and /or legal guardian. History taking and clinical examination was done and recorded in the form of a proforma. History included age, sex, duration of seizure, type of seizure, time, any predisposing factors, antecedent events if any, pork ingestion, contact with open case of tuberculosis etc. Detailed ,head to foot, examination including examination for any focal neurological deficit was done. Neuro imaging (mri) was obtained after stabilization.

In those imaging studies where no obvious visually detectable changes were found,hippocampal volumetry was done using these steps:

- Acquisition of MRI slices(coronal) and evaluation in a DICOM viewer(radiant) and exporting them in the form of JPEG image .

- Creating stacks of image slices IMAGE J software.
- Marking REGION OF INTEREST on the stacked image and measuring the area of it.
- Area is the multiplied with the number of slices stacked varying per viewer/and or patient
- Sum of these values per slice is used to calculate volume of 3D structure.
- The acquired data is entered into a MICROSOFT EXCEL sheet and analysed.

Hippocampal Volume calculation

Step 1 ImageJ software(version 1.33) was downloaded from http://www.rsb.info.nih.gov/ij/download.html.

Step 2 Stack creation- Relevant MRI slices were evaluated in the original viewer called RADIANT DICOM VIEWER. The software is downloadable for free. Every MRI slice has a distinctive cipher or number so as to be able to be set up in the information menu of the viewer,which matches a JPEG file. The opened in ImageJ to Create a stack using “Convert Images to Stack” function.

Step 3 Scale adjusting - subsequent to opening DICOM images in ImageJ, the scaling of the images is automatically corrected by the software, and volumetric analysis can be continued. However, in non-

DICOM viewers, the scale of the imported stack was adjusted by measuring the distance between two randomly chosen but clearly recognisable points on a slice in the original viewer using its measurement tool. Subsequently, the line between these points was traced on the corresponding slice and its distance set in ImageJ using the “Set scale” function .

Step 4 Region of Interest creation - On the MRI slices, the region of interest (ROI) pertinent for the study at hand is the hippocampus. The region of interest is selected using polygon selection tool with multiple clicks to outline the area.

Step 5 To calculate volume the area of ROI is multiplied by slice thickness for each and added together.giving the total volume of structure.

Marking Region of Interest

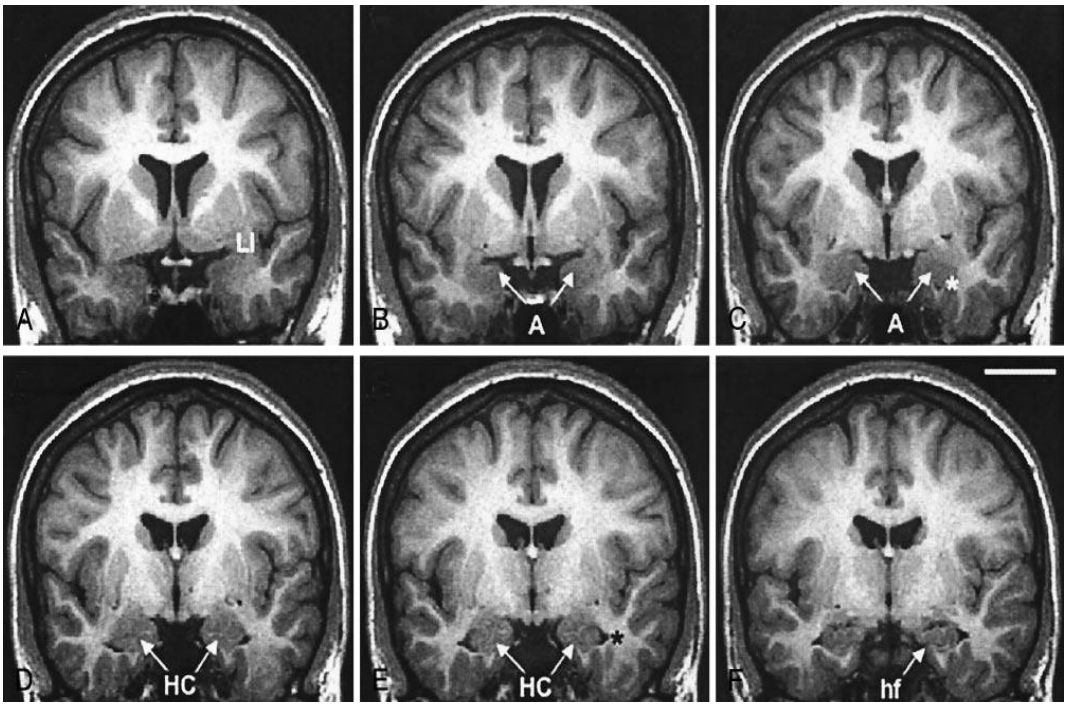


FIGURE A : most rostral where limen insulae is identified(li)

FIGURE B : first section where characteristic oval shaped AMYGDALA(A) is identifiable

FIGURE C : full extent of AMYGDALA where lateral ventricle is seen beneath(*)

FIGURE D : ROSTRAL HIPPOCAMPUS(HC) is seen

FIGURE E : HIPPOCAMPAL HEAD(HC) AND LATERAL VENTRICLE(*)

FIGURE F : HIPPOCAMPAL FISSURE (HF)

Statistical Analysis

Statistical analysis was done by using percentages, mean values, standard deviation, standard error, chi square tests.SPSS version 20 was used to analyse data. The level of significance used was 0.05 levels for the corresponding degree of freedom to draw the inference. A p-value < 0.05 was considered to be statistically significant and a p -value > 0.05 was considered to be not statistically significant.

OBSERVATION AND INFERENCES

OBSERVATION AND INFERENCES

Frequency Table

Age(1a)

Particulars	No.of respondents (n=56)	Percentage (100%)
12 to 20yrs	24	42.9
21 to 40yrs	19	33.9
41 to 60yrs	12	21.4
61yrs & above	1	1.8

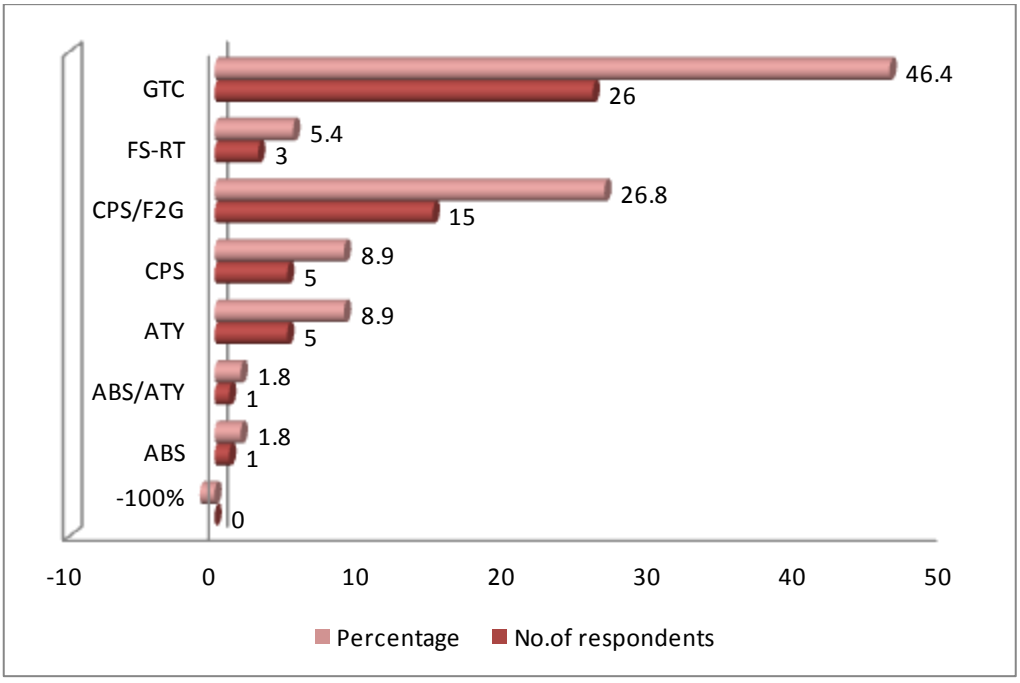
Sex (1b)

Particulars	No.of respondents (n=56)	Percentage (100%)
Male	26	46.4
Female	30	53.6

TOS(1c)

Particulars	No.of respondents (n=56)	Percentage (100%)
ABS	1	1.8
ABS/ATY	1	1.8
ATY	5	8.9
CPS	5	8.9
CPS/F2G	15	26.8
FS-RT	3	5.4
GTC	26	46.4

GRAPHICAL REPRESENTATION (1c)



FOCAL NEUROLOGICAL DEFICIT(1d)

Particulars	No.of respondents (n=56)	Percentage (100%)
No	46	82.1
Yes	10	17.9

LEVEL OF CONSCIUOSNESS(1e)

Particulars	No.of respondents (n=56)	Percentage (100%)
C	52	92.9
D	4	7.1

FEBRILE SEIZURES (1f)

Particulars	No.of respondents (n=56)	Percentage (100%)
No	52	92.9
Yes	4	7.1

DURATION OF SEIZURES (1g)

Particulars	No.of respondents (n=56)	Percentage (100%)
Below 1 month	15	26.8
13 to 24 months	19	33.9
25 months & above	22	39.3

PORK INGESTION (1h)

Particulars	No.of respondents (n=56)	Percentage (100%)
No	54	96.4
Yes	2	3.6

CT BRAIN(1i)

Particulars	No.of respondents (n=56)	Percentage (100%)
? SDH	1	1.8
G	6	10.7
Hydrocephalus	1	1.8
Hyperdensity	2	3.6
ICH	4	7.1
NS	28	50.0
PCA	4	7.1
REL	9	16.1
S	1	1.8

TB.CONTACT (1j)

Particulars	No.of respondents (n=56)	Percentage (100%)
No	53	94.6
Yes	3	5.4

MRI

VOLUMETRY(1k)

Particulars	No.of respondents (n=56)	Percentage (100%)
No	37	66.1
Yes	19	33.9

ECG (1l)

Particulars	No.of respondents (n=56)	Percentage (100%)
IVCD,TWC	1	1.8
LVH	3	5.4
NAD	42	75.0
PJP	4	7.1
RBBBB	2	3.6
RBBB/LEAD1 SIGN	1	1.8
SB	3	5.4

U.ALB(1m)

Particulars	No.of respondents (n=56)	Percentage (100%)
Nil	56	100

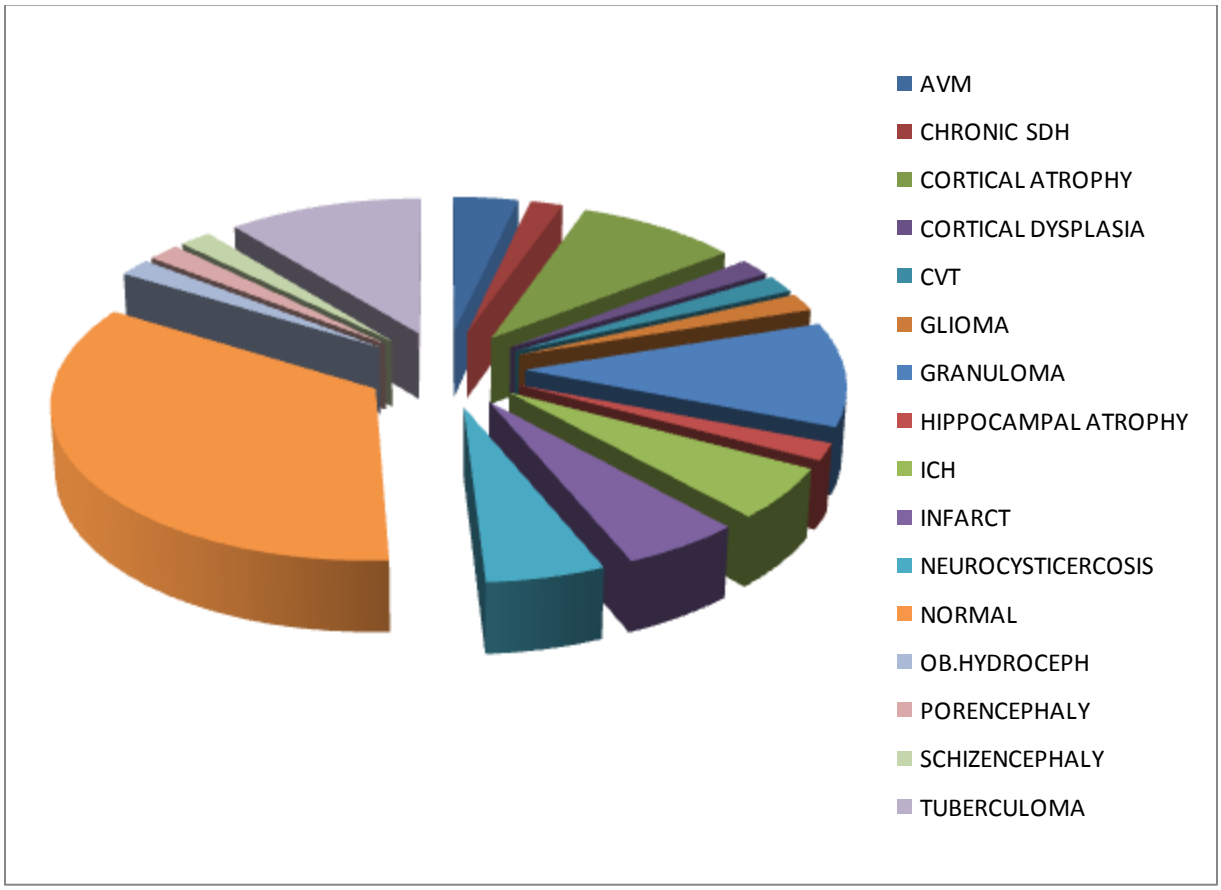
u.sugar (1n)

Particulars	No.of respondents (n=56)	Percentage (100%)
Nil	56	100

MRI result(1o)

Particulars	No.of respondents (n=56)	Percentage (100%)
AVM	2	4
CHRONIC SDH	1	2
CORTICAL ATROPHY	5	9
CORTICAL DYSPLASIA	1	2
CVT	1	2
GLIOMA	1	2
GRANULOMA	6	11
HIPPOCAMPAL ATROPHY	1	2
ICH	3	5
INFARCT	3	5
NEUROCYSTICERCOSIS	3	5
NORMAL	19	34
OB.HYDROCEPH	1	2
PORENCEPHALY	1	2
SCHIZENCEPHALY	1	2
TUBERCULOMA	6	11

ILLUSTRATION FOR TABLE 1o



Descriptive Statistics(2)

Item	Min.	Max.	Mean	S.D
Age	12	65	27.50	13.512
RHV	2.16	4.95	3.9179	.62346
LHV	2.22	4.91	3.9847	.69433
THV	.00	9.59	2.6812	3.83464
IHD(RHV-LHV)	-1.18	.88	-.0227	.34769
RBS	94	182	132.37	22.065
UREA	18	58	27.48	8.863
CREAT	.60	1.80	.9125	.20808
s.sodium	133	145	138.89	3.013
S.K+	3.40	4.40	3.8714	.26403

Paired Samples 't' Test(2a)

	Mean	S.D	Mean	S.D	t	df	Statistical inference
Pair 1							
RHV (n=19)	3.9179	.62346	-.0668	.60519	-.481	18	.636>0.05 Not Significant
LHV (n=19)	3.9847	.69433					
Pair 2							
RHV(n=19)	3.9179	.62346	-3.9847	.69433	-25.016	18	.000<0.05 Significant
THV(n=19)	7.9026	1.17275					
Pair 3							
LHV(n=19)	3.9847	.69433	-3.9179	.62346	-27.392	18	.000<0.05 Significant
THV(n=19)	7.9026	1.17275					

T-Test(2b)

Gender	Mean	S.D	Statistical inference
RHV			
Male (n=7)	4.0429	.49698	T=.657 Df=17 .520>0.05 Not Significant
Female (n=12)	3.8450	.69686	
LHV			
Male	4.1000	.67139	T=.542 Df=17 .595>0.05 Not Significant
Female	3.9175	.72775	
THV			
Male(n=26)	2.1923	3.72023	T=-.887 Df=54 .379>0.05 Not Significant
Female (n=30)	3.1050	3.94419	

T-Test(2c)

LEVEL OF CONCSIUOSNESS	**Mean**	**S.D**	**Statistical inference**
RHV			
C (n=17)	3.9241	.65574	T=.123 Df=17 .903>0.05 Not Significant
D (n=2)	3.8650	.33234	
LHV			
C (n=17)	4.0335	.71908	T=.888 Df=17 .387>0.05 Not Significant
D (n=2)	3.5700	.14142	
THV			
C (n=52)	2.6015	3.83177	T=-.557 Df=54 .580>0.05 Not Significant
D (n=4)	3.7175	4.29401	

Oneway ANOVA (2d)

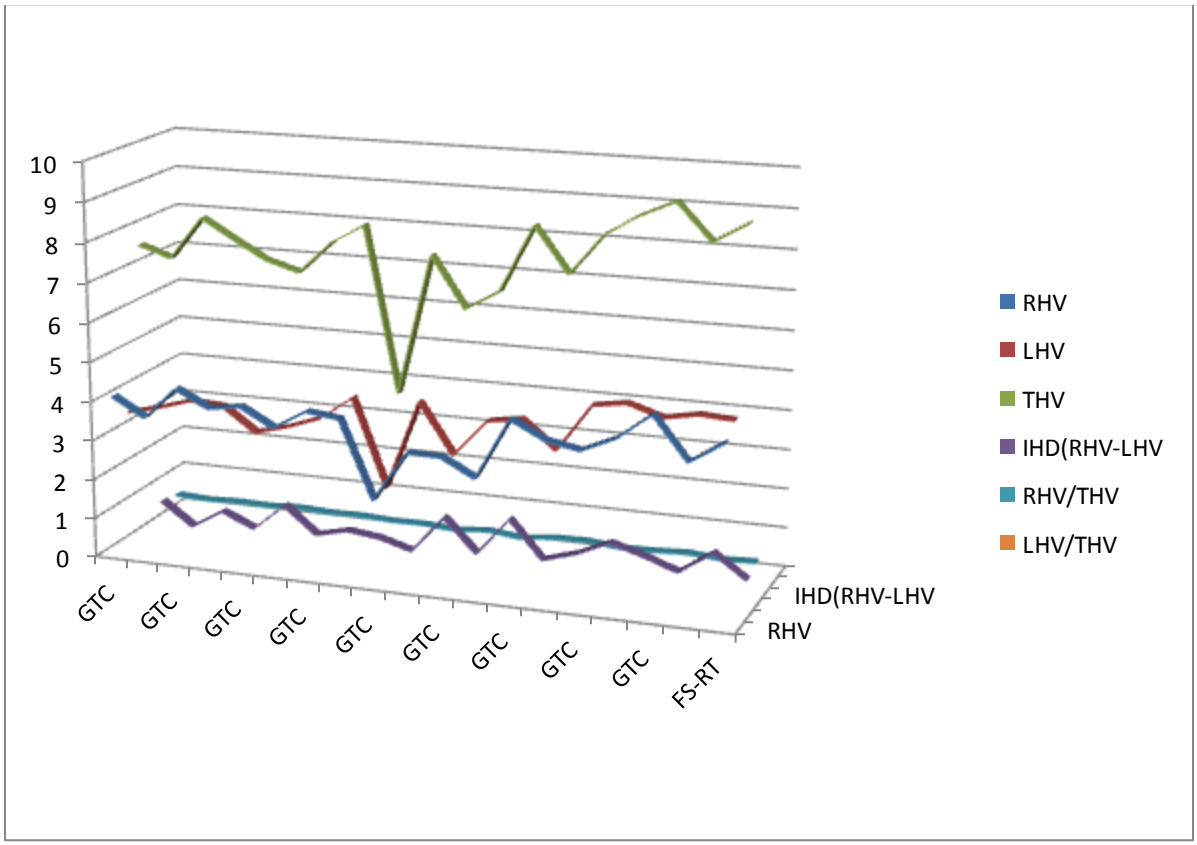
	Mean	S.D	SS	Df	MS	Statistical inference
RHV						
Between Groups			1.090	2	.545	F=1.476 .258>0.05 Not Significant
Below 1 month (n=5)	3.7420	.47378				
13 to 24 months (n=7)	3.7300	.77035				
25months & above (n=7)	4.2314	.49131				
Within Groups			5.907	16	.369	
LHV						
Between Groups			1.373	2	.687	F=1.504 .252>0.05 Not Significant
Below 1 month (n=5)	3.9480	.56220				
13 to 24 months (n=7)	3.6857	.81465				
25months & above (n=7)	4.3100	.58569				
Within Groups			7.304	16	.457	
THV						
Between Groups			.287	2	.143	F=.009 .991>0.05 Not Significant
Below 1 month (n=5)	2.5633	3.76753				

13 to 24 months (n=7)	2.7321	3.77287				
25months & above (n=7)	2.7177	4.10294				
Within Groups			808.460	53	15.254	
IHD(RHV-LHV)						
Between Groups			.061	2	.030	
Below 1 month (n=5)	-.0687	.45247				F=.244 .784>0.05 Not Significant
13 to 24 months (n=7)	.0163	.33312				
25months & above (n=7)	-.0250	.28655				
Within Groups			6.588	53	.124	

Paired Samples ‘t’ Test(2e)

	Mean	S.D	Mean	S.D	t	df	Statistical inference
Pair 1							
RHV (n=19)	3.9179	.62346	-.0668	.60519	-19.481	18	.006<0.05 Significant
LHV (n=19)	3.9847	.69433					

TOS to Hippocampal volumetry(2f)



Oneway ANOVA (2g)

Age	Mean	S.D	SS	Df	MS	Statistical inference
RHV						
Between Groups			.615	2	.308	F=.771 .479>0.05 Not Significant
12 to 20yrs (n=8)	3.9050	.46852				
21 to 40yrs (n=7)	4.1057	.57000				
41 to 60yrs (n=4)	3.6150	.98243				
Within Groups	.	.	6.381	16	.399	
LHV						
Between Groups			.314	2	.157	F=.300 .745>0.05 Not Significant
12 to 20yrs (n=8)	3.8913	.35167				
21 to 40yrs (n=7)	4.1529	.68232				
41 to 60yrs (n=4)	3.8775	1.25231				
Within Groups			8.364	16	.523	

Age vs THV(2h)

Age	Avg.THV
12-20	7.88
21-30	7.89
31-40	8.71
41-50	6.92

Correlations

		RHV	LHV	THV	IHDRHVLHV
RHV	Pearson Correlation	1	.583**	.877**	.362
	Sig. (2-tailed)		.009	.000	.128
	N	19	19	19	19
LHV	Pearson Correlation	.583**	1	.902**	-.547*
	Sig. (2-tailed)	.009		.000	.015
	N	19	19	19	19
THV	Pearson Correlation	.877**	.902**	1	-.132
	Sig. (2-tailed)	.000	.000		.591
	N	19	19	19	19
IHDRHVLHV	Pearson Correlation	.362	-.547*	-.132	1
	Sig. (2-tailed)	.128	.015	.591	
	N	19	19	19	19

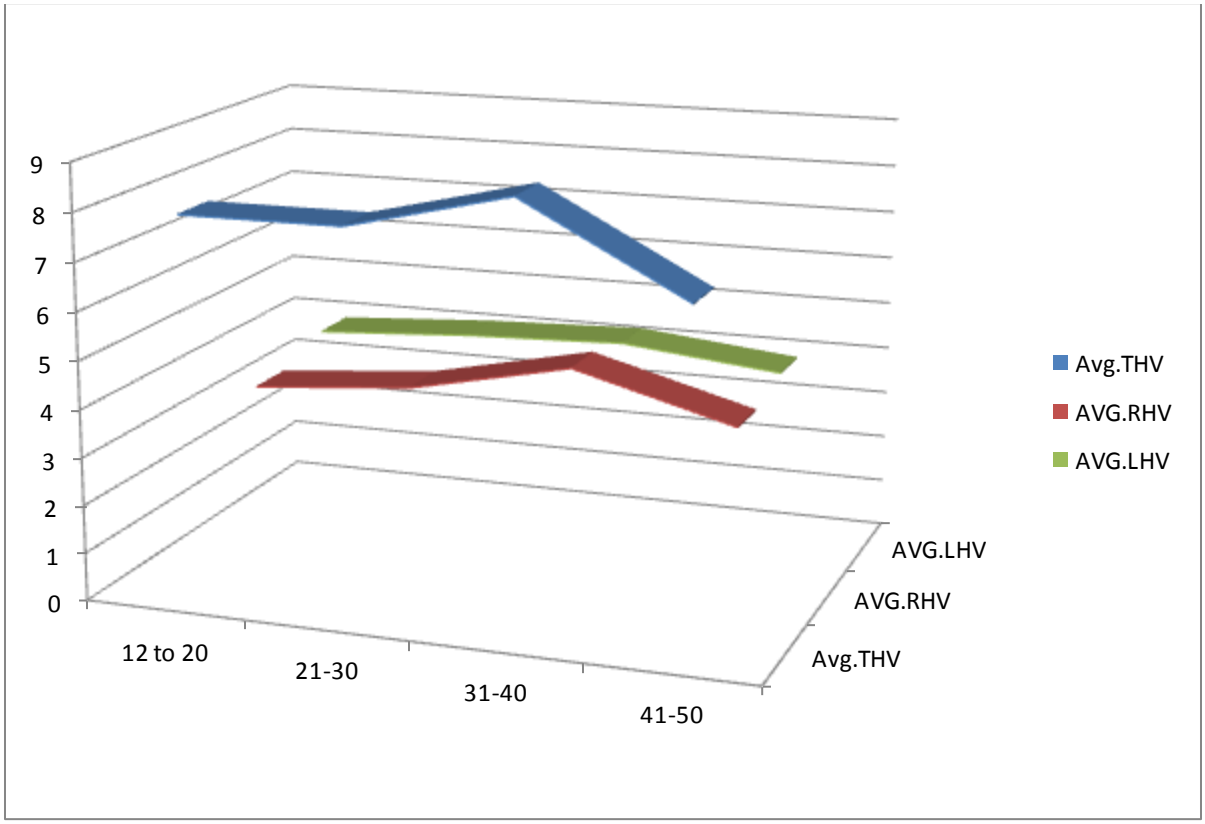
**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

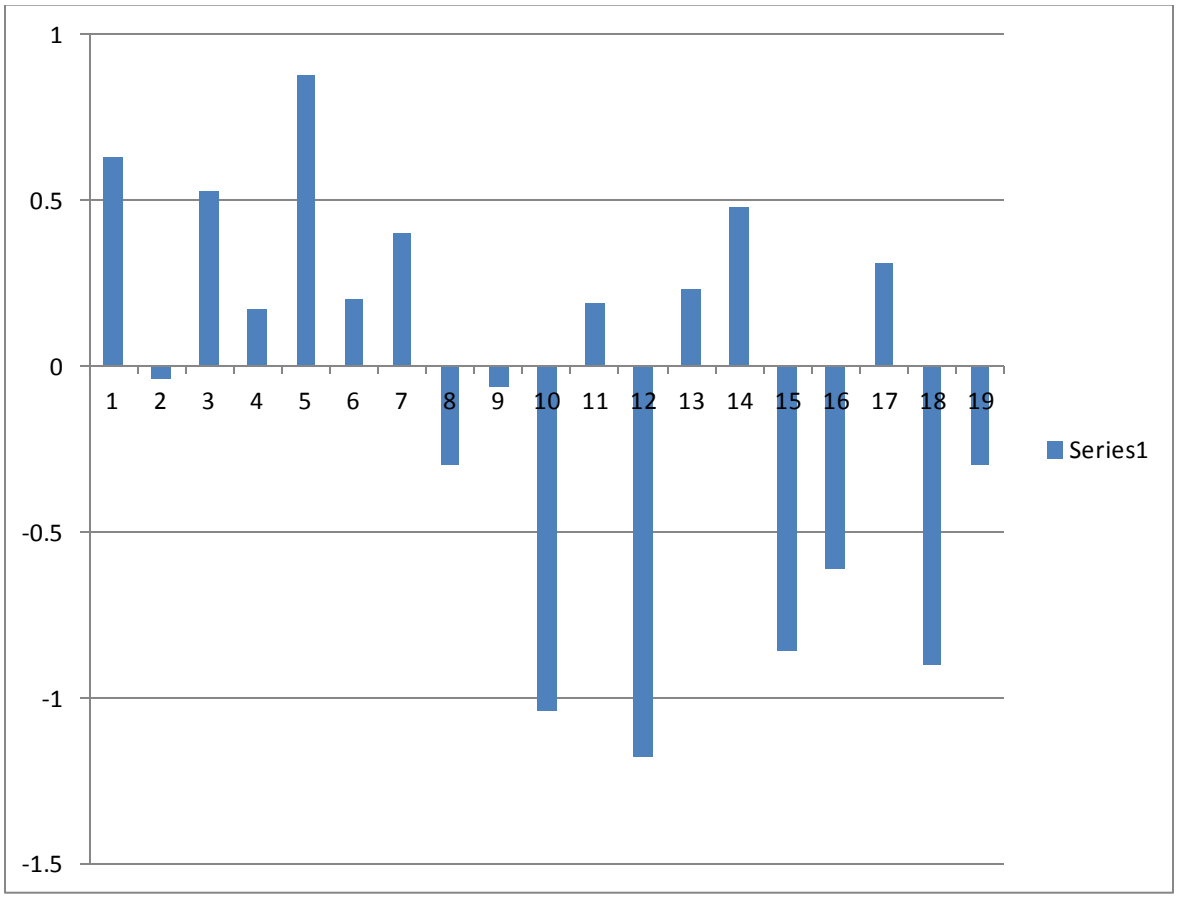
Relation between age hippocampal volume(2j)

Age	Avg.THV	AVG.RHV	AVG.LHV
12 to 20	7.88	3.49	3.89
21-30	7.89	3.79	4.1
31-40	8.71	4.52	4.22
41-50	6.92	3.61	3.87

Illustration(2j)



Variation in Interhippocampal difference(IHD)(2k)



IHD = RHV - LHV

DISCUSSION

This study was conducted to determine the various patterns of seizure and to correlate them with imaging studies, mainly MRI .The study population was 56 adults presenting to M.G.M GH with seizures. Metabolic causes of seizures have been ruled out with basic biochemical investigations.patients with diabetes,ckd have been excluded from the study.hippocampal volumetry was done for patients with normal imaging.

Seizure type

Approximation of distribution of patterns of seizure in adults was Gtcs(46.4%) followed by complex partial seizures with secondary generalization (26.8%),complex partial seizures (8.9 %),atypical seizures 8.9% ,absence seizures (3.6%) of these the predominant type of seizure in 12-20 age group is partial seizure(simple and complex with or without secondary generalization); 21-40 age group shows a predominance in gtcs; and 41 to 65 age group shows an equal prevalence of gtcs and partial seizures.absence seizure was reported only in 12-20 age group.

this is in concordance with literature reference[74]

Onset of seizure

Most of the seizure had their onset in the age group in 12-20 range(22),21-40 age range showed 16 cases , 10 cases had onset in the 41 to 60 age group ,7 cases had their onset in the less than 12 age group, and 1 case in the more than 60 age group. Most number of seizures manifest at the age range of 12 - 40 .this was also in relative concordance with previous studies and published material[74.]

Sex

There was a difference in preponderance as , of all the cases 53.6 % were females and 42.9 % were males focal deficit in those patients presenting with seizure 88% had no focal neurological deficit 12 % had focal deficit in the form of quadriparesis, hemiparesis and congenital lateral rectus palsy.

Febrile seizures

7.1% of the cases reported a history of febrile seizures in childhood of which one had MRI diagnosis of cortical atrophy,2 had normal MRI reports and NORMAL volumetry,1 was diagnosed as Tuberculoma.

Duration of seizure

In our study, 26.8% subjects had onset of seizure within one month; 33.9% between 13 and 24 months and 39.3% above 24 months. Of the subjects with duration of seizures more than 2 years, 6 had normal MRI study. 50% of these cases had IHD variability from the mean value of .49. 50% cases had a larger left hippocampal volume than right and the rest vice versa.

Biochemical parameters

Basic biochemical parameters (blood urea, serum creatinine, serum electrolytes, serum pottasium, urine albumin, blood sugar, urine sugar) were within normal limits in all cases with no history of diabetes mellitus, hypertension,chronic kidney disease. Thus, ruling out the possibilty of metabolic cause for seizure.

Electro cardio gram

ECG findings in these cases did not show any significant derangements to classify a type of seizure into one of its differentials as in syncope, stoke adams attack.

MRI findings

Among all the subjects ,34% had a normal mri report on conventional assessment, the rest had structural abnormalities as in tuberculoma (11%), granuloma (11%), premature cortical atrophy (9%), neurocysticercosis (5%), intracranial hemorrhage (5%), infrct (5%), arteriovenous malformations(4%), avm with hemorrhage (2 %),chronic sdh(2%),cvt(2%),glioma(2%), cortical dysplasia (2%), porencephaly (2%), schizencephaly(2%), Hippocampal atrophy (2%). Among 5 cases diagnosed as Tuberculoma , 3 patients gave the history of contact with a known case of tuberculosis. Of the 3 cases reported as neurocysticercosis,1 gave history of Pork ingestion . Hippocampal volumetry was done in patients with normal imaging findings (19 cases). There was a significant($p<.05$) difference between the mean left hippocampal volume(3.98) and mean right hippocampal volume(3.92) .this is in discordance with the study done by Edith V.Sullivan et al [74]where they found a larger right than left hippocampus. 2 cases showed significant reduction in hippocampal volume of the 2 cases one subject, age 48(RHV -2.16 cubic cm[45% reduction from mean], LHV – 2.22cubic cm [45% reduction from mean LHV],THV- 4.18cubic cm) presenting with gtcs with age at onset of 48. other was a 15 year old female (RHV – 2.97 cubic cm [25% reduction from mean LHV],LHV - 4.15 cubic cm ,THV – 7.12) mean RHV = 3.91 cubic cms, mean LHV =

3.98 cubic cms the mean Interhippocampal difference was .49 cubic cms ,8 cases showed a variability from this value, of which 3 cases had a larger RHV and 5 cases had a larger LHV ,2 cases showed a difference more than twice the mean Interhippocampal difference.

Type of seizure in cases with image negativity

Among the cases with normal MRI 14 cases presented with gtcs,2 with cps(with or without secondary generalization) ,1 had focal seizure ,1 had absence seizure and 1 with atypical seizure.

Age and Total Hippocampal volume(THV)

Relation between distribution of age and Total hippocampal volume showed a curvilinear function in that age range 12-20 showed a mean THV of 7.8,21 to 30 showed 7.89,31 to 40 showed 8.71 and 41 to 60 showed 6.92 this finding .this finding is in concordance with previous studies and meta analysis which also showed a curvilinear variability of Hippocampal size with age[76].

Limitations of the study

In this study,

i. Correlation with EEG is lacking
ii. Term of study is relatively short
iii. Study population needs to be larger
iv. Details of AED intake and duration of treatment could have given us insight about the intractable nature of seizures in the study group which would have given us an important clue for us to form a conclusion regarding causal relationship between seizure and hippocampal regression and vice versa.

SUMMARY

1. The most common type of seizure in adults is GTCS
2. The predominant type of seizure in
    a) 12-20 age group is partial seizure(simple and complex with or without secondary generalization);
    b) 21-40 age group shows a predominance in GTCS;
    c) 41 to 65 age group shows an equal prevalence of GTCS and Partial seizures.
    d) Absence seizure was reported only in 12-20 age group

3. Normal imaging in MRI is most commonly associated with GTCS type of seizure.
4. Focal and complex partial seizures are predominantly associated with neuroimaging abnormalities.
5. Most common MRI finding in SEIZURE patients is Normal study.
6. Every patient with epilepsy invariably needs an MRI for complete evaluation.
7. Tuberculomas are amongst the most common finding in IMAGE POSITIVE MRI results .
8. Hippocampal volumetry revealed gross volume regression in 2 subjects with one bilateral regression.

9. 8 cases out of the 19 with MRI NEGATIVE SEIZURES had significant variability in Interhippocampal volume difference from the mean IHD.
10. Correlation between IHD and duration of seizure is equivocal.
11. One subject showed a significant unilateral hippocampal regression on volumetry who may be a potential candidate for epilepsy surgery and needs further work up.
12. There is correlation between age and Total Hippocampal Volume with a peak Hippocampal volume in the 31-40 age range.

CONCLUSION AND RECOMMENDATIONS

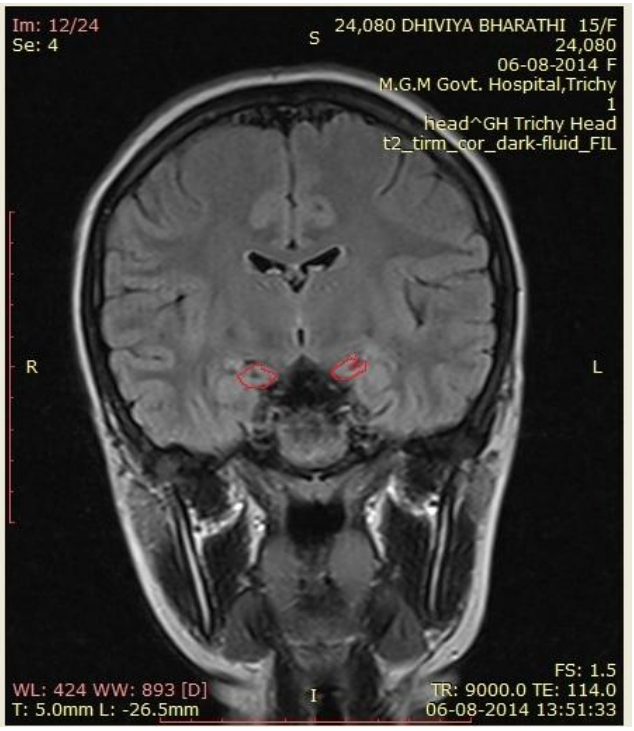
1. History and physical examination have no substitute.
2. MRI is more sensitive and specific for imaging in patients with seizure than CT .
3. Hippocampal volumetry is essential in patients with MRI negative seizures in setups where investigations like fMRI, SPECT and PET are unavailable.
4. More extensive studies with longer term and larger population of study are required before we can establish a clear set of guidelines.
5. Further investigation and information regarding an option of Epilepsy surgery should be offered to the patients with unilateral Hippocampal regression on volumetry as they may progress to Refractory Epilepsy.
6. Subjects with significant variability in Inter Hippocampal volume difference need regular follow up with imaging to rule out the development of Hippocampal regression.

ANNEXURES

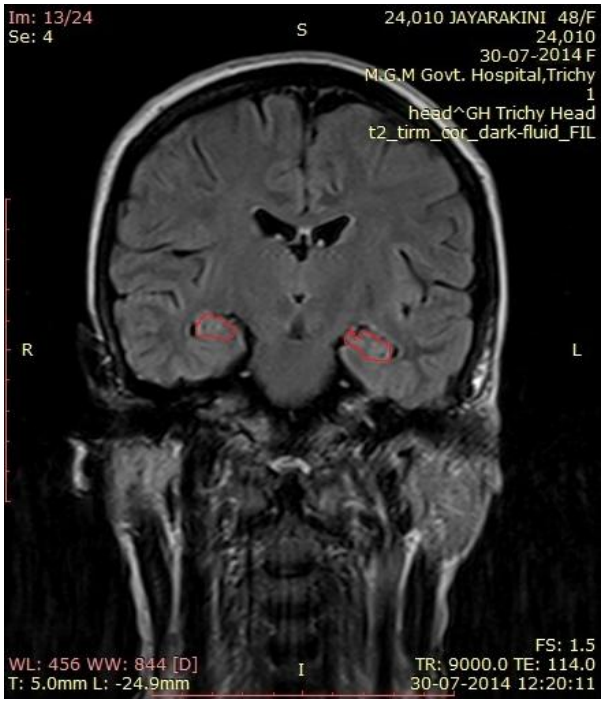
NEUROIMAGING

HIPPOCAMPAL REGRESSION

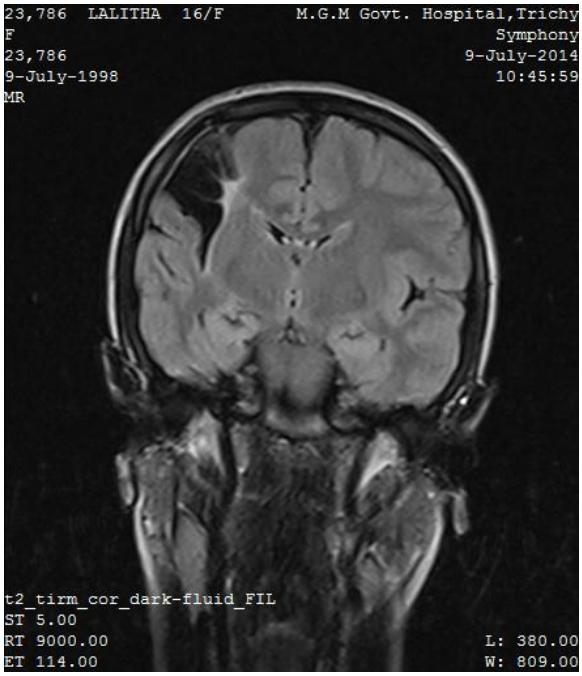
UNILATERAL(Right)



BILATERAL

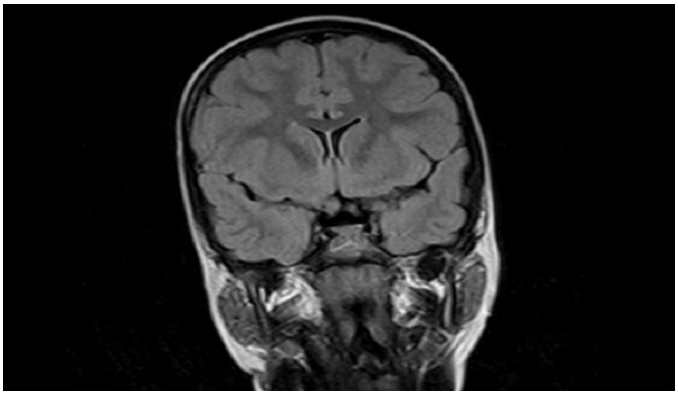


SCHIZENCEPHALY



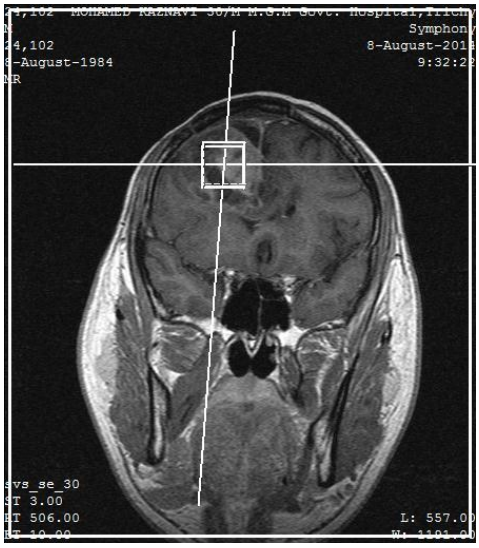
PORENCEPHALY

DIFFUSE CORTICAL ATROPHY

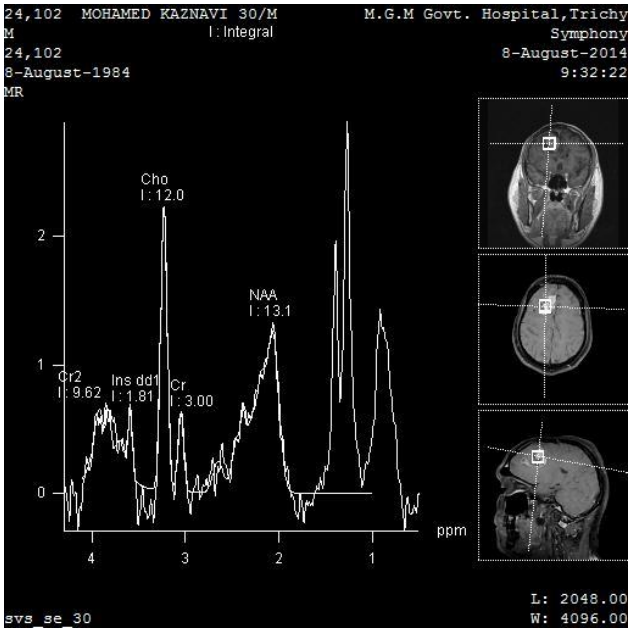


ICH with AVM

FRONTAL GLIOMA



MR SPECTROSCOPY



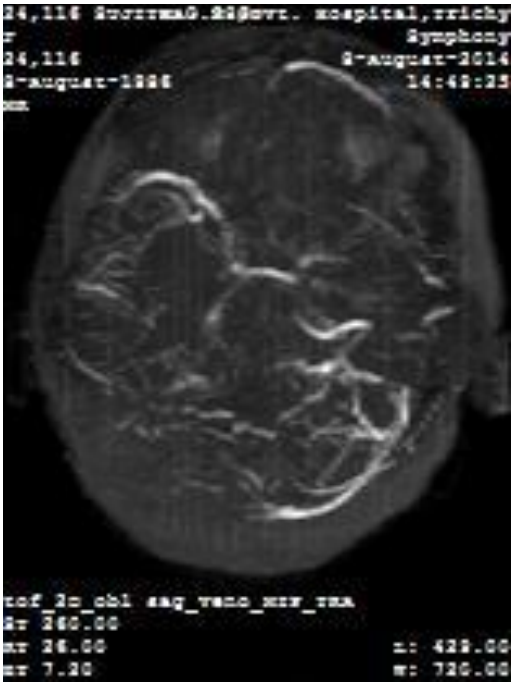
PREMATURE CORTICAL ATROPHY

TUBERCULOMA

NEUROCYSTICERCOSIS

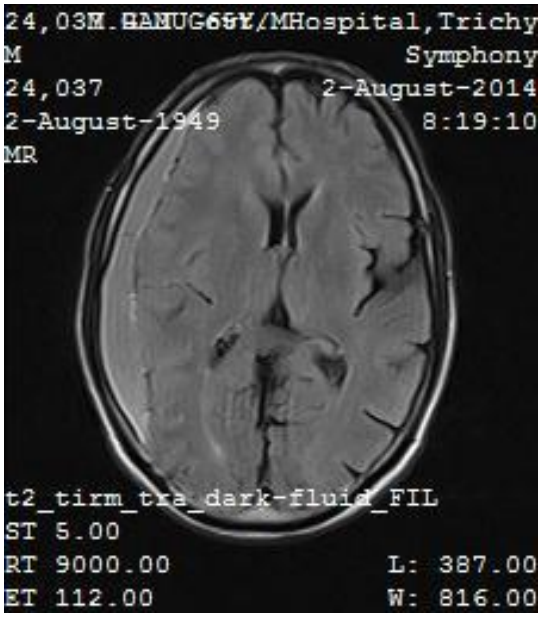
CVT WITH INFARCT

CVT



GRANULOMA

SDH



MULTIPLE GRANULOMA

TUBERCULOMA

MASTER CHART

sl.no:	age	AAO	sex	TOS	FD	LOC	FS	DOS	PI	CT	TB	VOLUMETRY	RHV	LHV	THV	IHD(RHV-LHV	RBS	ECG	UREA	CREAT	UA	US	SOD	POT	mr diagnosis
1/23798	12	12	F	GTC	NO	D	NO	3MONTHS	NO	NS	NO	YES	4.1	3.47	7.57	0.63	98	NAD	19	0.9	NIL	nil	137	3.6	NORMAL
2/23799	12	12	M	GTC	NO	D	NO	2WEEKS	NO	NS	NO	YES	3.63	3.67	7.3	-0.04	104	NAD	21	1	NIL	nil	140	3.4	NORMAL
3/23812	40	38	F	GTC	YES	D	NO	2YRS	NO	PCA	NO	NO			0	0	108	RBBB	24	0.8	NIL	nil	142	4.1	CORTICAL ATROPHY
4/23829	25	22	M	GTC	NO	C	NO	3YRS	NO	PCA	NO	NO			0	0	112	NAD	20	0.8	NIL	nil	136	3.8	CORTICAL TROPHY
5/23834	30	1	F	GTC	NO	C	NO	30YRS	NO	NS	NO	YES	4.45	3.92	8.37	0.53	107	NAD	28	1	NIL	nil	139	4.2	NORMAL
6/23845	12	12	M	ABS	NO	C	NO	1WEEK	NO	NS	NO	YES	4.03	3.86	7.89	0.17	121	NAD	19	0.8	NIL	nil	141	3.6	NORMAL
7/23867	23	15	F	CPS/F2G	NO	C	NO	8YRS	NO	G	NO	NO			0	0	125	NAD	26	0.9	NIL	nil	134	3.7	GRANULOMA
8/23870	33	33	F	GTC	NO	C	NO	1MONTH	NO	NS	NO	YES	4.16	3.28	7.44	0.88	136	NAD	24	0.8	NIL	nil	136	4	NORMAL
9/23929	26	25	M	GTC	NO	C	NO	1YEAR	NO	HYDROCEPHA	NO	NO			0	0	117	SB	36	1.1	NIL	nil	133	3.9	OB.HYDROCEPH
										LUS															
10/23935	19	19	M	ABS/ATY	NO	C	NO	1MONTH	NO	G	NO	NO			0	0	124	PJP	28	1.1	NIL	nil	137	3.4	GRANULOMA
11/23962	18	5	F	GTC	NO	C	YES	13 YRS	NO	PCA	NO	NO			0	0	118	NAD	19	0.6	NIL	nil	138	4.1	CORTICAL ATROPHY
12/23981	12	12	F	CPS/F2G	NO	C	NO	3MONTHS	NO	NS	NO	YES	3.7	3.5	7.2	0.2	138	NAD	22	0.7	NIL	nil	141	3.7	NORMAL
13/23982	14	8	F	CPS/F2G	YES	C	NO	6YRS	YES	G	NO	NO			0	0	118	NAD	19	0.9	NIL	nil	137	4.3	GRANULOMA
14/23983	16	16	F	GTC	NO	C	NO	4MONTH	NO	NS	NO	YES	4.2	3.8	8	0.4	124	NAD	18	0.7	NIL	nil	142	4	NORMAL
15/23992	21	21	M	ATY	NO	C	NO	8MONTHS	YES	REL	NO	NO			0	0	138	NAD	21	0.9	NIL	nil	141	3.7	NEUROCYSTCERCOSIS
16/23994	19	17	F	GTC	NO	D	NO	2YEARS	NO	NS	NO	NO			0	0	128	PJP	26	0.6	NIL	nil	142	3.8	CORTICAL DYSPLASIA
17/24001	14	4	F	GTC	NO	C	YES	10YEARS	NO	NS	NO	YES	4.1	4.4	8.5	-0.3	120	PJP	21	0.7	NIL	nil	144	4	NORMAL
18/24010	48	48	F	GTC	YES	C	NO	4MONTHS	NO	NS	NO	YES	2.16	2.22	4.38	-0.06	148	NAD	28	0.9	NIL	nil	136	4.2	NORMAL
19/24037	65	65	M	CPS/F2G	YES	C	NO	8MONTHS	NO	?SDH	NO	NO			0	0	168	SB	46	1.2	NIL	nil	139	4.1	CHRONIC SDH
20/24040	29	29	F	GTC	NO	C	NO	2MONTHS	NO	NS	NO	YES	3.42	4.46	7.88	-1.04	130	NAD	38	0.9	NIL	nil	142	3.7	NORMAL

21/2 4062	24	20	M	GTC	NO	C	NO	4YRS	NO	NS	NO	YES	3.41	3.22	6.63	0.19	108	NAD	21	0.8	NIL	nil	134	3.9	NORMAL
22/2 4102	30	21	M	CPS/F2G	YES	C	NO	9YEARS	NO	NS	NO	NO			0	0	142	NAD	48	1	NIL	nil	136	4	GLIOMA
23/2 4109	42	42	F	CPS/F2G	NO	C	NO	2MONTHS	NO	REL	NO	NO			0	0	178	IVCD, TWC	40	1.1	NIL	nil	136	4.2	TUBERCULOMA
24/2 4116	28	28	F	GTC	NO	C	NO	6MONTHS	NO	NS	NO	NO			0	0	118	NAD	24	0.9	NIL	nil	134	4	CVT
25/2 4080	15	15	F	CPS/F2G	NO	C	NO	1WEEK	NO	NS	NO	YES	2.97	4.15	7.12	-1.18	106	PJP	26	1	NIL	nil	140	3.6	NORMAL
26/2 4114	17	17	F	CPS/F2G	NO	C	NO	3DAYS	NO	G	NO	NO			0	0	136	NAD	22	0.6	NIL	nil	144	3.5	GRANULOMA
27/2 4104	13	7	F	GTC	NO	C	YES	6YRS	NO	NS	NO	YES	4.51	4.28	8.79	0.23	117	NAD	18	0.9	NIL	nil	135	3.9	NORMAL
28/2 4148	19	19	M	CPS/F2G	YES	C	NO	5DAYS	NO	ICH	NO	NO			0	0	114	SB	58	1.8	NIL	nil	137	3.8	ICH/AVM
29/2 4191	14	14	M	CPS/F2G	NO	C	YES	8MONTHS	NO	REL	YES	NO			0	0	134	NAD	36	0.8	NIL	nil	142	3.6	TUBERCULOMA
30/2 4138	42	41	M	ATY	NO	C	NO	1YEAR	NO	NS	NO	YES	4.08	3.6	7.68	0.48	156	LVH	34	1.4	NIL	nil	139	4.1	NORMAL
31/2 4225	45	45	F	GTC	NO	C	NO	2MONTHS	NO	NS	NO	NO			0	0	152	lrbbb	36	1.2	nil	nil	141	3.7	INFARCT
32/2 4235	42	42	F	GTC	YES	C	NO	1MONTH	NO	NS	NO	YES	3.92	4.78	8.7	-0.86	132	NAD	28	0.8	nil	nil	136	4.1	NORMAL
33/2 4259	21	15	F	FS-RT	NO	C	NO	6YRS	NO	PCA	NO	NO			0	0	148	NAD	26	1.1	nil	nil	138	3.8	PORENCEPHALY
34/2 4247	45	43	M	GTC	NO	C	NO	2YEARS	NO	NS	NO	YES	4.3	4.91	9.21	-0.61	168	NAD	20	0.9	nil	nil	136	4	NORMAL
35/2 4300	40	40	M	GTC	YES	C	NO	1WEEK	NO	ICH	NO	NO			0	0	124	NAD	30	0.8	NIL	nil	140	4.2	ICH
36/2 4325	14	14	M	CPS/F2G	NO	C	NO	1MONTH	NO	REL	NO	NO			0	0	94	NAD	21	0.8	NIL	nil	136	3.5	NEUROCYSTICERCOSIS
37/2 4336	33	30	M	GTC	NO	C	NO	3YRS	NO	NS	NO	YES	4.95	4.64	9.59	0.31	128	NAD	24	0.8	NIL	nil	143	3.7	NORMAL
38/2 4341	26	22	M	GTC	NO	C	NO	4YRS	NO	NS	NO	YES	3.9	4.8	8.7	-0.9	142	NAD	20	0.9	NIL	nil	141	3.8	NORMAL
39/2 4338	31	28	M	ATY	NO	C	NO	3YRS	NO	HYPERDENSITY	NO	NO			0	0	158	NAD	24	0.9	NIL	nil	139	3.6	AVM
40/2 4339	13	2	M	ATY	NO	C	NO	11YRS	NO	HYPER	NO	NO			0	0	118	NA	18	0.6	NIL	nil	135	4.1	AV

										NSITY														
41/24356	33	33	F	FS-RT	NO	C	NO	2MONTHS	NO	NS	NO	YES	4.45	4.75	9.2	-0.3	166	NAD	28	1	NIL	nil	144	4.2	NORMAL
42/24357	25	25	F	GTC	NO	C	NO	2DAYS	NO	NS	NO	NO			0	0	134	NAD	48	1.2	NIL	nil	136	3.6	INFARCT
43/23855	19	19	M	CPS/F2G	NO	C	NO	2MONTHS	NO	REL	YES	NO			0	0	174	NAD	26	1.1	NIL	nil	139	3.9	TUBERCULOMA
44/24223	55	52	M	GTC	NO	C	NO	3YEARS	NO	NS	NO	NO			0	0	114	RBBB/LAD1SIGN	42	1	NIL	nil	142	4.1	CORTICAL ATROPHY
45/24387	19	19	M	CPS/F2G	NO	C	NO	4MONTHS	NO	REL	NO	NO			0	0	128	NAD	28	0.8	NIL	nil	141	3.5	TUBERCULOMA
46/23838	45	40	F	GTC	NO	C	NO	5YEARS	NO	NS	NO	NO			0	0	146	NAD	24	0.7	NIL	nil	138	4.2	HIPPOCAMPAL ATROPHY
47/24388	45	45	M	CPS	YES	C	NO	4DAYS	NO	NS	NO	NO			0	0	174	NAD	38	1.1	NIL	nil	136	3.8	INFARCT
48/24362	19	19	F	CPS/F2	YES	C	NO	1WEEK	NO	REL	YES	NO			0	0	130	NA	21	0.8	NIL	nil	140	4.4	TUBERCULOMA

				G													D							
49/23786	16	8	F	CPS	NO	C	NO	8YRS	NO	S	NO	NO			0	0	118	NAD	19	0.8	NIL	nil	142	3.7	SCIZENCEPHALY
50/24389	50	50	F	FS-RT	NO	C	NO	1DAY	NO	ICH	NO	NO			0	0	182	LVH	34	1.1	NIL	nil	137	4	ICH
51/24383	43	33	F	CPS	NO	C	NO	10YEARS	NO	G	NO	NO			0	0	126	NAD	24	1	NIL	nil	145	3.6	GRANULOMA
52/33067	28	12	M	GTC	NO	C	NO	16YEARS	NO	NS	NO	NO			0	0	108	NAD	34	0.9	NIL	nil	137	4.2	CORTICAL ATROPHY
53/23866	17	17	F	CPS/F2G	NO	C	NO	3MONTHS	NO	G	NO	NO			0	0	134	NAD	22	0.8	NIL	nil	141	3.4	GRANULOMA
54/24891	14	14	M	ATY	NO	C	NO	2MONTHS	NO	REL	NO	NO			0	0	178	NAD	24	0.7	NIL	nil	143	4.1	NEUROCYSTICERCOSIS
55/24994	16	16	M	CPS	NO	C	NO	5MONTHS	NO	REL	NO	NO			0	0	108	NAD	24	0.8	NIL	nil	140	4.2	TUBERCULOMA
56/24394	54	54	F	CPS	NO	C	NO	3DAYS	NO	ICH	NO	NO			0	0	138	LVH	26	0.9	NIL	nil	138	3.5	ICH

PROFORMA

NAME :

DATE OF ADMISSION :

AGE :

PATIENT NUMBER :

INFORMANT :

ADDRESS :

CONVULSIONS

DATE

PLACE

NO. OF EPISODES

DURATION

TYPE OF SEIZURE

NATURE OF SEIZURE

TONIC-CLONIC/TONIC/CLONIC/ATONIC/ABSENCE

(TYPICAL/ATYPICAL)

FOCAL/FOCAL WITH SECONDARY GENERALIZATION

ASSOCIATED FACTORS

PRODROME/AURA/AUTONOMIC PHENOMENA/LOC/BLADDER

AND BOWEL INCONTINENCE/ FROTHING/ AUTOMATISMS/

ACTIVATION

POST ICTAL DEFICIT

TODD'S PALSY

RECOVERY FROM ICTAL PHASE

SPONTANEOUS/WITH MEDICATIONS

PREVIOUS EPISODES

H/O FEBRILE SEIZURES

FAMILY HISTORY

COMORBIDITIES

EXAMINATION

GENERAL EXAMINATION VITALS

NEUROLOGICAL EXAMINATION

HIGHER MENTAL FUNCTIONS

LEVEL OF CONSCIOUSNESS

GLASGOW COMA SCALE

E - /V- /M - -

ORIENTATION

SPEECH\MEMORY

HANDEDNESSILLUSION/DELUSION/HALLUCINATION

CRANIAL NERVE EXAMINATION

	RIGHT	LEFT
OLFACTORY		
SMELL		
OPTIC		
LIGHT REFLEX		
VISUAL ACUITY		
VISUAL FIELDS		
COLOUR VISION		
FUNDUSCOPY		

3RD ,4TH ,6TH *PTOSIS* *PUPIL(RTL,ACCOMODATION)* *POSITION OF EYEBALL* *RANGE OF MOVEMENTS* *SMOOTH PURSUIT* *NYSTAGMUS* *SACCADES*		
TRIGEMINAL ***MOTOR*** *MASSETER* *TEMPORALIS* *PTERYGOIDS* *JAWJERK* ***ENSORY*** *SENSATION OVER FACE* *CORNEAL RELEX*		

FACIAL ***MOTOR*** *FRONTALIS* *ORBICULARIS OCCULI* *ORBICULARIS ORIS* ***SENSORY*** *TASTE* *SECRETOMOTOR*		
VESTIBULOCOCHLEAR ***AUDITORY*** *RINNES* *WEBER'S* *ABSOLUTE BONE CONDUCTION* ***VESTIBULAR*** *VERTIGO* *CALORIC STIMULATION*		

GLOSSOPHARYNGEAL *TASTE(POSTERIOR THIRD)* *PHARYNGEAL REFLEX*		
VAGUS *PALATAL REFLEX* *LARYNX(VOICE)*		
CRANIAL AND SPINAL ACCESSORY STERNOCLEIDOMASTOID TRAPEZIUS		
HYPOGLOSSAL ***TONGUE*** WASTING FASCICULATION TREMOR DEVIATION		

MOTOR SYSTEM *NUTRITION* *POSTURE* *TONE* *POWER* *CO-ORDINATION*		
DEEP TENDON REFLEXES *BICEPS* *TRICEPS* *SUPINATOR* *KNEE* *ANKLE* *SUPERFICIAL REFLEXES* *ABDOMINAL* *CREMASTERIC* *BULBOCAVERNOSUS* *ANAL* *PRIMITIVE REFLEXES* *POUT* *GRASP* *PALMOMENTAL* *PLANTAR*		

SENSORY SYSTEM *CRUDE TOUCH* *FINE TOUCH* *PAIN* *TEMPERATURE* *JOINT POSITION SENSE* *VIBRATION*		
GAIT AND STANCE *WALKING PATTERN* *ROMBERG'S SIGN* *HEEL TO TOE* *HEEL WALKING* *TOE WALKING*		
CEREBELLAR SYSTEM *TITUBATION* *HYPOTONIA* *REBOUND PHENOMENON* *NYSTAGMUS* *PENDULAR KNEE JERK* *DISDIACHOKINESIS* *FINGER NOSE TEST* *HEEL KNEE TEST*		

AUTONOMIC NERVOUS SYSTEM

BOWEL

BLADDER

SWEAT

RESTING TACHYCARDIA

SUPINE BLOOD PRESSURE

STANDING BLOOD PRESSURE

SUSTAINED HAND GRIP

DEEP BREATH

OTHER SYSTEMS

INVESTIGATIONS

RANDOM BLOOD SUGAR

BLOOD UREA

SERUM CREATININE

SERUM ELECTROLYTES

URINE ALBUMIN/SUGAR

ECG – ALL LEADS

IMAGING

COMPUTED TOMOGRAPHY

Infectious

Genetic-congenital

Traumatic

Degenerative

Toxic

Metabolic

Inherited

Acquired

Neoplastic

Inflammatory-immune

MAGNETIC RESONANCE IMAGING

Infectious

Genetic-congenital

Traumatic

Degenerative

Toxic

Metabolic

Inherited

Acquired

Neoplastic

Inflammatory-immune

ADDITIONAL REMARKS PERTAINING SPECIFICALLY TO CASE

ABBREVIATIONS

MRI – Magnetic resonance imaging

CT - Computerised axial tomography

TOS – Type of SEIZURE

AAO – Age at onset

DOS – Duration of seizure

PI – Pork ingestion

TB – Tuberculosis

RHV – Right Hippocampal volume

LHV- Left Hippocampal Volume

THV – Total Hippocampal volume

IHD – Inter Hippocampal Volume Difference

VOL- Volumetry

RBS- Random blood sugar

UA – Urine Albumin

US – Urine Sugar

CREAT – Serum Creatinine

FD – Focal Neurological deficit

FS – Febrile seizure history

CA – cortical atrophy

CD – Cortical Dysplasia

HA – Hippocampal Atrophy

REL – Ring Enhancing Lesion

NS – Normal study

ICH – Intracranial hemorrhage

GTC – Generalized tonic clonic seizure

CPS/F2G – Complex partial seizure with secondary generalization

ABS – Absence seizure

ATY – Atypical seizure

FS-RT – Focal seizure right side

S- Schizencephaly

P-Porencephaly

AED – Antiepileptic drugs

PET – Positron Emission Tomography

SPECT – Single Photon Emmision Computed Tomography

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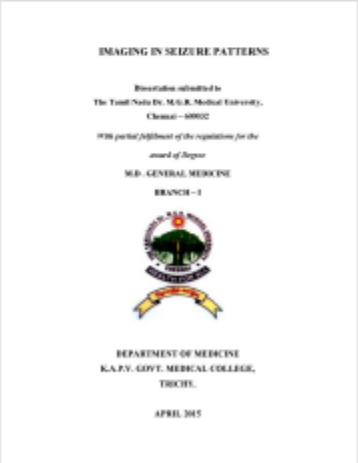

IMAGING IN SEIZURE PATTERNS

Dissertation submitted to
The Tamil Nadu Dr. M.G.R. Medical University,
Chennai – 600032

With partial fulfilment of the regulations for the
award of Degree

M.D. - GENERAL MEDICINE

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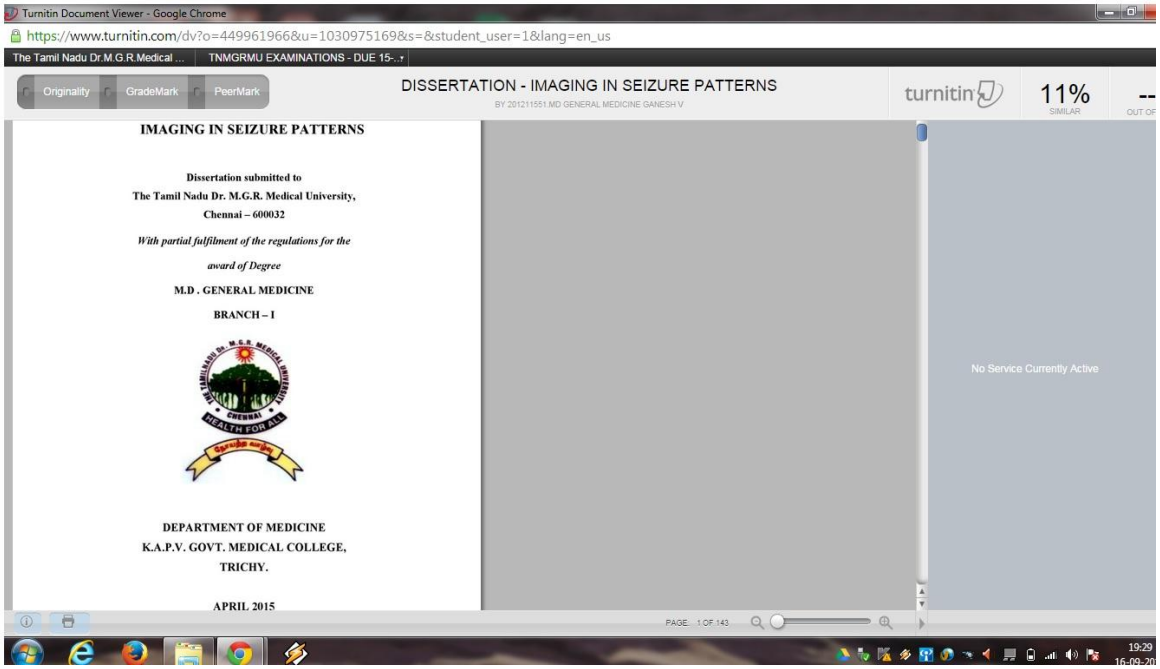
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19:29